

Suelen Queiroz

Treaty Occupational Toxicology

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Suelen Queiroz

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Suelen Queiroz

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Suelen Queiroz





*To my parents
Jose Helio Queiroz
and Maria dos Navegantes Queiroz,
who taught me respect and love for medicine and not
allow themselves to kill the chance events of life.*

Preface

The occupational health and chemical agents in Sao Paulo and Brazil, which is faced in day to day lives of millions of Brazilians is not considered by many as under the relevant legal expertise. Usually those who express themselves so do not know science and contempt is the weapon used. There is, indeed, one side of the active center and a disadvantage to claim the right to tax insured by the Federal Constitution and others, in passive, an autonomous government agency to federal (INSS) which holds a state monopoly. Perhaps part of the work there to answer the questions and the sentiment expressed at the trial with a scientific orientation.

It is a message of courage of Honor Dr Sr ° °: Irineu Antonio Pedrotti, the author of "occupational diseases or work":

"The love and dedication to duty has no barriers. You can plant a tree and cultivating it with affection and sometimes it will sacrifice a day or flowers, fruit, or shadow to treat. "

The value of work

The world today is in a full search process for maximum yield and maximum cost. Such a goal is due to the fact that the demand by developing and underdeveloped countries, the pursuit of global economic control by the developed countries. Of course, that interest is related to the well being of humankind, since the state has the primary goal, the society. To achieve these goals, countries have to have an essential factor, technology.

This factor brings positive economic benefits, since there is an investment in the binomial - Man Machine. However, it is necessary to consider that this factor may contribute to a result contrary to that expected by the state, because there will be a direct influence on the working environment of man. Therefore, it is necessary something that will protect human labor, then there is the concept of security.

In 1700, was published in Italy, a book whose author was a doctor named Bernardino Ramazzini, which had repercussions throughout the world due to its importance. In this work, Ramazzini describes fifty different professions and diseases related to them. It introduced a new concept by Ramazzini, "What is your occupation?". Today we might interpret this question as follows: "Say what your job, to say the risks that you are subject." For this important work, Bernardino Ramazzini was known as the "Father of Medicine of Labor."

At the time of publication of this book, professional activities were still handmade, being carried out by small numbers of workers and, consequently, cases of occupational diseases were few, or little interest has arisen regarding the problems cited in the work of Ramazzini.

In the eighteenth century, then there is almost a century later, in England, the Industrial Revolution, a movement that would change the entire design in relation to work performed, and accidents and occupational diseases stemmed from them. The first factories were located close to watercourses, because the machines were driven by hydraulic power, owing to its location, it had a shortage of workers. With the advent of steam power, factories could be located in large cities where the manpower was achieved more easily.

The manufacturing operations become simplified by machinery introduced in production. The tasks to be performed by the worker were repetitive, which led to an increasing number of accidents. Coupled with the fact mentioned above, there was no criterion for the recruitment of skilled labor, where men, women and even children were selected without any initial examination as to the health and physical development or other

human factors. The demand for manpower was so unscrupulous that these children were purchased from destitute parents, coming to be accepted by a retarded child group for each of twelve healthy children. The number of accidents increased dramatically, where the death of children was often caused by poorly designed machines, which offered no security.

As production was first, there were limits on hours of work, and gas burners used for nighttime.

In the work environment were caused by poor noise machines, high temperatures, due to lack of ventilation, poor lighting, etc.. those factors that contributed to the high number of accidents, because until the work orders in production were not heard by the employee due to the high noise level.

In the process of 'working man's activity logs, so by means of work, processing the work object, desired from the beginning. This process is void in the product. Your product is a value in use, a natural material adapted to human needs through processing the form. The work is united with his goal, worked. Before all the work is a process between man and nature, a process in which man, by her own actions, mediates, regulates and controls your metabolism with nature. He faces the same natural materials as a natural force. He sets in motion natural forces belonging to his physicality, arms and legs, head and hands in order to appropriate the natural material in a form useful for your own life. By working through this movement, on the Nature external to it and modify it, modify it, while his own nature. He develops the powers it dormant and may subject the game of his forces to his own domain .. . We assume the job in a way that belongs exclusively to man. A spider conducts operations that resemble those of the weaver, and the bee ashamed over a human architect to build the combs of their beehives. But what distinguishes, in advance, the worst architect from the best of bees is that he built the comb on his head, before you build it in wax. At the end of the working process, you get a result that already existed at the beginning of this work in the imagination .. . He not only performs a transformation of the natural form of matter, held at the same time, the natural matter, seuobjetivo, he knows he determines the law as to the nature and mode of his activity and which has to subordinate his will. And this subordination is not an isolated act. Besides the efforts of agencies that work is required the will directed to an end, which manifests as attention throughout the course of work .. . The simple elements of the work process are activities geared to one end or the work itself, its object and its means. "(Marx, Capital, I, 1 section III, chap. V)

This treatise is dedicated to the teaching and dissemination of

information that constitutes the formation of knowledge of occupational diseases, only through security measures and effective enforcement of the PPE (personal protective equipment) and regular occupational medical examinations is that we can reduce the numbers accidents and chronic diseases caused by occupational poisoning.

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Dedication

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Contents

PREFACE	6
THE VALUE OF LABOR	7
ACKNOWLEDGEMENTS	10
DEDICATION	11
INTRODUCTION	15
OATH OF AREA MEDICAL	30
HISTORY AND CONCEPTS OF TOXICOLOGY	32
CONCEPT OF TOXICOLOGY	35
CHAPTER 1 - Poisoning	37
CHAPTER 2 - OCCUPATIONAL DISEASES	48
CHAPTER 3 - MAIN occupational dermatitis	84
REFERENCES	108
ANNEXES	110
CHAPTER 4 - LEGAL SUPPORT	125
CHAPTER 5 - GENERAL OCCUPATIONAL TUMORS	129
CHAPTER 6 - THE ILLNESS OF WORKERS AND ITS RELATION TO WORK	136
CHAPTER 7 - BASIS FOR CONTROL TECHNIQUES OF RISK FACTORS AND TO IMPROVE OF ENVIRONMENTAL AND WORKING CONDITIONS	139
CHAPTER 8 - IDENTIFICATION AND CONTROL OF RISK FACTORS IN THE PERSPECTIVE OF HYGIENE LABOR AND ERGONOMICS	152
CHAPTER 9 - 5.1 DIAGNOSIS OF A DISEASE RELATED TO WORK AND REMOVAL OF WORK	157
CHAPTER 10 - RELATED DISEASES LABOR INFECTIOUS DISEASES RELATED WORK	162
CHAPTER 11 - WORK-RELATED DISEASES PROCEDURES MANUAL FOR HEALTH SERVICES	200
CHAPTER 12 - Neoplasm (tumor) WORK-RELATED	238

CHAPTER 13 - Blood and blood forming organs	
RELATED WORK	244
CHAPTER 14 - Aplastic ANEMIA TREATMENT AND	
OTHER CONDUITS	263
CHAPTER 15 - DISEASES ENDOCRINE, NUTRITIONAL	
AND METABOLIC WORK-RELATED	279
CHAPTER 16 - MENTAL DISORDERS AND	
BEHAVIOUR RELATED WORK	292
CHAPTER 17 - PREVENTION	299
REFERENCES	343
TABLES	348

INTRODUCTION

Since the ancient Greco-Roman, work was already seen as a factor generating and modifying the conditions of living, getting sick and dying men. Works of Hippocrates, Pliny, Galen and others called attention to the importance of the environment, seasonality, type of job and social position in determining the production of disease.

With the social reality of those times, when nations were enslaving other nations subdued in war, these reports were unlikely to have the stamp of social protest. *De Re Metallica*, the work of Georg Bauer (Georgius Agricola), 1556, refers to lung disease in miners, with interesting description of symptoms now attributed to silicosis, asthma and Agricultural called miners. Already Paracelsus, in 1567, it also describes diseases of miners in the region of Bohemia and mercury poisoning. And in 1700, the extraordinary work of Bernardino Ramazzini, a doctor who worked in the region of Modena in Italy, and with an impressive vision clinic for that time where there were no major propaedeutic, describes diseases occurring in over fifty occupations. In his book *From Morbis Artificum Diatriba* can find, besides the sharpness of the comments, a subtle criticism of manners. Because of the importance of his work, received from posterity the title of father of Occupational Medicine. Ramazzini, in anticipation of some basic concepts of Social Medicine, emphasized the importance of studying the relationship between the health status of a given population and their living conditions, which were, he said, depending on the social situation (Rosen, 1994). And actually, if you look in the background the political and social panorama of contemporary Europe, we find the view accepted by many Ramazzini, adding the idea that social life and everything that she was referring (as working conditions and health) should be at the service of the State, setting up there as one of elements of a doctrinal system, which was later called mercantilism or cameralism. Returning to the references of diseases related to work, remember Morgan in the *Treaty of Pathology*, 1761 apud MENDES (1980) observed and emphasized the occupation item, the reporting of all cases reported. PERCIVAL POTT, in 1776, and made the first detailed references to scrotal cancer in chimney sweeps, which actually was formed in March as the initial studies of the relationship between cancer and work (cited in Mendes, 1994).

In 1700, Ramazzini later c the "Father of Occupational Medicine", published his work, "*De Morbis Artificum Diatriba*" a series of work-

related diseases. With progress, new industrial processes have emerged and with them came new duties or occupations. It has become increasingly imperative to the existence of services that take care of health and welfare of workers. Adverse environmental conditions of work have favored the emergence of occupational diseases and the occurrence of accidents whose consequences are widely known. Across Europe, mainly in Germany but also in France and England, extends the doctrine of Medicine of the State, based ideologues such as Petty says, "... A healthy population is synonymous with opulence and power." He lived the concern with the growing urbanization, with issues of food for the people, sanitation, major epidemics (Foucault, 1987). The medicine is beginning to become collective, urban, social (Mendes, 1994). And it is precisely here that a new fact arises that would modify an entire global economic system, with consequences for social and health of European populations: the INDUSTRIAL REVOLUTION. Historically, the second makes clear ROSEN (1994), one of the factors responsible for the development of the modern world and the organization and actions of modern public health was the rise of an industrial economy that, for most authors, has its distinctive period between 1760 and 1850. Still living a feudal model of the Middle Ages, but with a growing movement of urbanization, industrialization of modern Britain starts, and the plants have settled mainly in urban areas. The craftsmanship, where the man was holding the whole process gives rise to an industrial process with profound social changes.

INDUSTRIAL REVOLUTION with a new situation arises: the work indoors, sometimes confined to what was called factories. The rural exodus, urban issues of sanitation and poverty were joined by another big problem: the poor working conditions (and environment) by changing the pattern of illness among workers who have suffered accidents and diseases in developing industrial areas, such as , European typhus (then called fever of plants). The majority of the workforce were women and children who suffered abuse by various agents, arising from the process and / or work environment. In 1831, C. Thackrar Turner, an English physician, in his book "The effects of arts, crafts and professions and the civil state and the life habits the heath and longevity," revealed the deplorable living and working in Leeds, England. Concern about the strength Working with economic losses prompted the intervention of governments in the factories. And we got to the early nineteenth century with the presence of doctors in factories (an emblematic example of Dr. Robert Baker, England, quoted by MENDES, 1980) and emergence of the first public health laws that strongly addressed the issue of health workers

(Factory Act , 1833, for example). The Occupational Medicine had its first milestone there. In the late nineteenth century was perceived a new era: the knowledge of scientific medicine, based on germ theory, amounted to recognition of the concepts of social medicine, where issues such as housing, sanitation, and others come in as co- determining factors in the genesis of the disease process. The model of medical services within the company has spread to several countries in Europe and other continents, along with the industrialization process, and assumed an important role in controlling the workforce through increased productivity and regulation of absenteeism (MENDES & DIAS, 1991).

We arrived at the beginning of the twentieth century with the world around with big changes. The Marxist ideology, socialism and communism, in opposition to capitalism and the First World War, the result of imperialism still inherited from the past century. All these facts, as he explains (HOBBSAWUN, 1995), produced deep changes in political and social landscape of the world. The process of industrialization and increasing urbanization have changed the landscape of the capital-labor ratio. The emerging trade union movement began to express the social control that the workforce needed. At the same time, new technologies, to incorporate new work processes, generated risks culminating in work accidents and occupational diseases. As we know, the end of last century and the beginning of this, were marked by great inventions, and the incorporation of this new collection of science and technology was not without damage. The dynamics of capital-labor ratio has changed, while keeping the final binomial: exploiter and exploited, capitalist and worker. The Occupational Medicine found that in the early twentieth century, has the perspective of medicine of the body, individual and organic, structured under the figure of the doctor's work as agent and, through empirical instruments, acted on its object, man worker with a clinical-therapeutic approach, in which at most is analyzing the microenvironment of work and the action of certain pathogenic agents (DIAS, 1994; Tambellini, 1993).

This model also proved to be insufficient by scientific reductionism and conceptual. The working man had his demands Biological yes, but also psychological and social, in this aspect, the industrial revolution came to bring that Leavell & Clark (1976) mental illness at worker.

In the early twentieth century, with the expansion and consolidation of the model began with the industrial revolution and the transnationalization of the economy, the need for measures and common parameters, such as regulation and organization of the work, which

standardize the countries producing goods industrialized. So it is, that was created the International Labour Organisation in 1919. This organization has already recognized, in their first meetings, the existence of occupational diseases. In turn, the capitalist model created transformed the relations between man and nature, among human beings, with work and with society. Came the scientific organization of work, Taylorism and Fordism, converting the worker subject to object, and assisted by modern theories of Directors, which had as purpose, although not exclusive, productivity. And therefore, the conquest of the market. The sciences have evolved in turn, setting up new fields of knowledge, especially pure and applied chemistry, engineering, social sciences and the incorporation of the dimension of psychoanalysis. Developed the first concepts of Industrial Hygiene, Ergonomics and strengthened to Engineering Safety. Similarly, in the field of Public Health, Schools began to be created, such as John Hopkins, of Pittsburgh, with an emphasis on preventive medicine as prominent figures who had Leavell & Clark. All this has set a new model based on multidisciplinary and multiprofessional, Occupational Health, who was born under the aegis of Public Health with a much broader view than the original model of Occupational Medicine. It should be noted that this has not disappeared, but was expanded by adding up the body of knowledge to knowledge embedded in other disciplines and other professions. Thus:

"The Occupational Health occurs mostly in large companies, with mapping of multi-and interdisciplinary, with the organization of multi-professional teams gradually, and the emphasis on industrial hygiene, reflecting the historical origins of medical services and the prominence of the industry in industrialized. "(Mendes, 1991).

The Occupational Health going to give a rational response, scientific, health problems for certain processes and work environments and through the Toxicology and parameters established as tolerance limits, it was attempted to quantify the response or resistance of the working man to factors occupational risk.

Even today, in major industrialized countries, the model is hegemonic Occupational Health, as an effective protective legislation for workers and the environment is combined with equally efficient action from the regulatory authorities (the example we have the Occupational Safety Health Administration on-OSHA and National Institute for Occupational Health and Safety, NIOSH, the United States of America). But even then, by virtue of the social movements of the 60s, where he discussed the model of society since even the intrinsic meaning of work, he felt the need for greater participation of workers and society as a

whole, the discussion the major issues pertaining to the area. In Europe, where they swarmed renewal movements broke out in Italy of the 70s, a movement of workers demanding greater participation in matters of health and safety, which resulted in changes to legislation such as the involvement of unions in monitoring environments work, the right to information (risks, environmental commitment, technological changes) and, finally, significant improvement in conditions and labor relations. This was the Italian labor movement. And changes in legislation on health and safety of workers kept happening in several countries, and the movement that began in Italy, arrived in Latin America, where political and social turmoil and the thirst for change led to germinate, based on health reform and democratizing the struggles that virtually all the countries of Central and South America is waging this context that the subject area Occupational Health was set up and framed in a context of profound changes in work processes that began in the 70s, and whose hallmark was a transnationalization of the economies where industries have moved to the Third World, especially those for causing damage to health or the environment, like pesticides, asbestos and lead.

Also the automation to computerization, outsourcing, when deciding remarkable transformations in the organization and work process, their impact on workers and their health. The Occupational Health also emerged as a new approach to protection of men and women, in light of the pressure of capital. The works of Laurell & Noriega-incorporated markedly this thread strength and influence of researchers and health workers throughout Latin America will help to determine the object of the worker, as the study of health-disease process of human groups from the perspective of the work. Was delineated a field under construction within the Public Health and the theoretical and methodological assumptions that Tambellini (1993) and MENDES (1994) expressed as a break with the hegemonic conception of establishing a causal link between the disease and a specific agent and avoid the extreme opposite of social determinism unique. Hence the theoretical conceptual Tambellini (1985) "Occupational Health is the area of knowledge and application technique that gives an account of understanding the multiple factors that affect the health of workers and their families, regardless of the sources they come from, the consequences of action of these factors on this population (diseases) and the varied ways of acting on these conditions ... "

From all that can be seen from these and other authors trying to draw an outline history and theory to the field worker's health, it is clear the role of the worker himself as a social actor, dynamic, suffering and

responding to the pressures of capital, and developing its own mechanisms of social control to a new time and form of organization of the work process. In summary, Occupational Health is defined as a set of theoretical practices developed by interdisciplinary and interagency actors situated in different places and different social bonds by a common vision (MINAYO-GOMEZ & Thedim, 1997). It is noted that the Occupational Health was consolidated in Latin America revisiting its own model, which comes 90 years to more pragmatic and less ideological, paradigmatic questioning some guidelines that delineated their practice more intense in the early '80s.

2. The second issue health-labor in Brazil

In the short history of Brazil, only five centuries, the way it structured and economically the country has also determined the type of relationship with work. The mineral extraction for which the knowledge and subsequent occupation of the Brazilian interior, the feudal agrarian model, the large estates of the planters and later of the colonels, the exploitation of native Indians, enslaved and sometimes also the years of black slavery, determined that - similarly to what happened in ancient times of ancient Egypt, Greece and Rome - the legwork was devoid of value attribute, and any disease processes that involve, as occupational diseases and work accidents, trivialized all the minor granted to labor deprived of any rights of citizenship.

With the abolition of slavery at the end of last century and the arrival of European immigrants earlier this century, Brazil begins his first major outbreak Industrial nearly one hundred years of delay. The great inventions of the late nineteenth century, the industrial revolution steam engine and textile industry, besides political, social and economical world, their impact on Brazil republic. The medicine then showed natural concern with major epidemics as well, playing the French social model of medicine, special care cemeteries, slaughterhouses, hospitals, prisons and factories. MENDES (1980) cites studies on the subject like MENDONÇA in 1850 who wrote "Of the snuff and cigar factories." And the model of industrial development in no way differed from that lived in England many years ago, primitive plants, with no minimum standards of hygiene, employing cheap labor, women and children in poor working conditions.

And we got to the early twentieth century, with profound changes in the labor-capital relations in the country, and indeed, in any form of organization and systematization of the work process, emphasizing the liberal movements of organizing workers, such as unions, basic socialist. To regulate the growth and to maintain the principle of power that amalgamates the Brazils, growing state control, similar to European

mercantilism. Thereafter, in the early twentieth century, we now have two references: the external influences, doctrinal, coming from Europe and the United States, and internally, the echo of the political and profound social changes in the country. Earlier this century, doctors of the National School of Medicine, Red Beach, protesting against the reality of the factories in Brazil. At the Congress of the young Republic of Brazil, there is the proposal in 1904, which grant social security benefits to workers injured at work, a constant, then the federal capital to Rio de Janeiro. (Mendes, 1980) Thereafter, with the greatest influence of North American medicine and public health schools such as Johns Hopkins, the teaching of hygiene at work came to be included in courses for health workers and later in medical courses in Brazil. Contradictorily. was in a period of totalitarian government (a dictatorship of the Estado Novo), emerged the Consolidation of Labor Laws - CLT, which grouped and systematized labor laws, representing remarkable progress of the legal point of view. It was also a period marked by growth and affirmation of the union movement in Brazil. Updates of the Law of Work Accidents ensued (FALEIROS, 1992). The technical and scientific development of Occupational Medicine, mainly in Rio de Janeiro and Sao Paulo passed by the existence of entities like the SESP Services (Public Health Specialist) and SESI (Social Service of Industry). Also in medical training, disciplines such as Occupational Medicine were included in the curricula of most medical schools in the country. It was created by the Brazilian Association of Occupational Medicine (ABMT), headquartered in Rio de Janeiro. Professionals such as Daphnes de Soto, Talita Tudor, Bernardo Bedrikow and Diogo Pupo Nogueira, among others, were prominent figures in the Occupational Health at the time. Proliferating medical services companies, asserting itself as the labor market also interesting for the country's doctors, especially those with training in Public Health (health workers) or in Preventive Medicine.

The 60 met Brazil in a new political crisis that culminated in the military coup of March 31, 1964. Brazil, also the former dictatorship of the Estado Novo, with Vargas, the decades between 30 to 50, started to live a restricted period of democratic freedoms at the same time that - keeping the logic of totalitarianism - the government tried to disciplinary the question of work, applying laws and reforms. Social Security Institutes of unified categories, not just to organize the policy of social benefits, but also to weaken the union movement. The Labor Department also consolidated its shares, intervening decisively in security and occupational health. In general, acatávamos technical recommendations of the International Labour Organisation (ILO) and later the Joint ILO /

WHO Committee and tuned it in our legislation. The jingoism of the beginning of military government, gave rise to attitudes such as the nationalization of insurance of occupational accidents in 1966, the revival of old slogans like "oil is ours" campaign for 200 miles (sovereignty of the coast). It was the third great industrial boom in the country, was the "Brazilian miracle", with the start of construction of works, such as Trans, Rio-Niteroi Bridge, football stadiums, dams, etc... Financed with internal and external indebtedness. The rapid pace of such works transformed the fields into battlefields, where workers were dying every day. In 1968 the world was true revolution of values, with the backdrop of the ideological and political dualism: x capitalism socialism. The tension of the Cold War, the threat of atomic holocaust, the failure of wars like Vietnam, but a real revolution in habits (ecological and peace movements, etc...) Probably had its heyday in 1968, and movements democratizing and libertarians have spread across Europe (Hobsbawm, 1995). Brazil, a country of young population markedly in that period, was influenced by such movements, and had its own history of suffering and revolt against the military dictatorship years in determining a period known as the year of lead or terror. The 70s came to meet the country dealing with such conflicts, political instability own periods of exception. The alternative to reducing accidents at work found by the military regime in the early '70s, due to the high rates of accidents, was the legal requirement for companies to hire skilled professionals (occupational physicians, nurses or nursing assistants labor, engineers and safety experts), thus creating Specialized Services in Safety Engineering and Occupational Medicine - SESMTs-sized according to the degree of risk and the number of employees of companies (Bonciani, 1994). The creation of such services was already recommended by the ILO since 1959, but was emphasized in Brazil in 70 years. It was a technical model tied to the business sector. Brazil's government was concerned exclusively with workplace accidents or by the economic repercussions (severe damage to public) or by the emphasis in all media: "Brazil, the world champion of workplace accidents." Little or no attention was given to the said illnesses. It is interesting to reproduce from a discourse of Arnaldo Prieto, Minister of Labour, in 1976, and also mentioned by Bonciani (1994):

"For this, we turn our attention to 1974, when the total of occupational injuries reached a figure of 1. 796. 761, with an average of 5. 891 accidents per working day, and that resulted in 3 totals. 373 permanently disabled (...) causing losses of eight (8) billion cruises. "

There was need for intervention measures, therefore. The academic level, the School of Public Health at USP (Universidade de Sao

Paulo) created the Department of Environmental Health, which housed an area of Occupational Health. By that time, many medical courses had also this area in their curricula. To prepare technical expertise in required number, the government established the Jorge Depart Figueiredo Foundation for Occupational Health and Safety - FUNDACENTRO Agency of the Ministry of Labor also affects the search. Graduate courses in Occupational Health and Safety Engineering have spread from north to south, usually in partnership with universities. Also on the legal profession, Government has extended and modified in Chapter V of the Labor Code, which deals with medicine and occupational safety, to create the Regulatory Norms (BRASIL. Ministry of Labour. 214) in June 1978, adopting quantitative assessment of environmental risks and limits of tolerance within the scope of Occupational Health, keeping the social security legislation, accident, with characteristics of a medicine practice, and on an individual level, considering only insured workers, or formerly engaged in the labor market (MENDES & DIAS, 1991) .

At the end of the 70 appeared in Brazil, two movements in the health field, with different elements within an early process of democratization of the country and of great importance. The first is called the Health Movement, which, based on the principles of the Conference of Alma-Ata (1978) and in its own internal struggle for citizenship rights in Brazil, including the access to health, conceived the Brazilian Health Reform Proposal seeking comprehensive care and overcoming the dichotomous model - preventive medicine, curative medicine (AUGUSTO, 1995).

The second movement is the Trade Union Movement, which did not by chance began at ABC, from the great strikes of 1978 in the automobile, and that spread through much of the country from there. (LACAZ, 1994). With the reorganization of the union movement, was introduced into the health issue on the agendas for discussion and claims, following at least there, the Italian workers' model from the 60s and 70s. In 1978, the Commission was established Inter-Health, Labor and that eventually became the Inter Department of Studies and Research in Health and Work Environments-DIESAT, which would support the important role trade unions in discussing health issues and work, trying to overcome welfare, inheritance of the Estado Novo (LACAZ, 1994). In parallel, several unions have structured specific boards, for the treatment of health concerns of workers. The union has added technical aspect that also was active in the health reform process in Brazil, others from academia, especially preventive medicine departments of universities (Tambellini, 1993). The unions, as the Center of workers (CUT) to create

the INSS had an important role in the so-called social control of the health issue at work. Was then launched one of the pillars of this new area, the Occupational Health, coming to fill a gap left by the previous models, of Occupational Medicine classical Occupational Health, who proved insufficient as well put MENDES & DAY (1991), because: Occupational Health, who proved to be insufficient, as well put MENDES & DAY (1991), because:

- A. is grounded in the mechanism;
- B. not performs a true interdisciplinary, towards the integration of knowledge in favor of the employee;
- C. the training of human resources, knowledge production and intervention technology, do not keep pace of transformation of work processes.

This new area, the Occupational Health, took the flags: the right to know, refusal to work in situations of risk, health is not exchangeable for cash, including health and environmental clauses in collective bargaining agreements, the actions of completeness occupational health, recognition of the knowledge workers' participation in the management of health services, consensual validation, homogenous groups of risk, incorporation of epidemiology as a tool for recognition of risk and harm to health and the environment (ODDONE, 1986). International agencies have also influenced the genesis of Occupational Health: the Pan American Sanitary Shop (PAHO) launched the document "Program of Action for Health of Workers" in 1983, and the Joint ILO / WHO launched the Convention 161 and Recommendation 171 (Service Health). Brazil, living in the said period for reopening or re-democratization, saw in Public Health, a new model with values from the health reform and nuclei of preventive medicine, the Occupational Health Program. The Occupational Health Program, which began to be created in several Brazilian states, from the early 80s, had his real moment of ideological creation with the VIII National Health Conference in 1986, an event that marked the philosophical principles SUS. So much so, that was convened for that same year the First National Conference on Occupational Health, which, by bringing an unprecedented, trade unionists, experts in the field of health and related fields, universities and community in general, successfully launched the foundations for a new path. The constitutional reform of 1988, defining citizenship rights as health and work, marked a move already in the New Republic said in a moment of political transition to democracy, to confirm the role of the State accountable for decent health care for workers and people in general.

In 1990 Brazil enacted the Organic Law of Health, 8. 080, which

constituted the reference came from the NHS and, indeed, not as a government initiative, but as a reflection of the struggles for reform health, which had the marks to VIII National Health Conference and the First National Conference on Occupational Health. 080 decisively address the issue of health worker in its Article 6 conceptualizing it as:

"... A set of activities that is intended, through epidemiological monitoring and health surveillance, promotion and protection of health workers, and seeks the recovery and rehabilitation of workers subjected to the risks and problems arising in working conditions ".

In 1991, the Ministry of Health, through its Division of Health Protection of Workers (DIPSAT), the new area created from the National Secretariat of Health Surveillance, promoted the National Seminar on Occupational Health, bringing together all the states of Federation, represented mostly by engineers or managers, Occupational Health Program, which was extensively, discussed the operationalization of the Act 8. . As a product of that seminar, I was scheduled the National Seminar on the injured worker, to be held in November 1991. In 1991, the Ministry of Welfare has published the new Costing and Benefits Act (8. 212 and 8. 213) with some significant advances in the security issue, such as stability to the injured worker.

An important achievement during the first half of the nineties was the establishment of the Commission on Occupational Health, whose report of November 1993 contained the principles of joint action from government agencies for the health of the worker. Progressed in the country the actions of environmental intervention aimed at protecting the health of the worker. Until 1993, were 161 Programs Occupational Health organized or being organized in Brazil (Dias, 1994)? This vitality could be found in the Second National Conference on Occupational Health held in March 1994, preceded by a pre-conference in nearly all Brazilian states and had about a thousand delegates, civil servants, representatives of trade unions, professional associations , technical advisors and academic organizations (AUGUSTO, 1995).

In December 1994, the Ministry of Health with the aim of encouraging the actions of the worker's health in states and municipalities, and forward the deliberations of the Second National Conference on Occupational Health, presented the Operational Standard for Occupational Health SUS (NOST). Aspects such as information system on occupational health, preparation of human resources, finance and several others were discussed in this document, which is proposed to be a guideline, so that in all regions of the country the practice of comprehensive health care workers are perform the desired quality.

In the same period, the Labor Department changed several regulatory standards that prevailed virtually intact since the Ordinance 3. How relevant aspects of the new NR 7, establishing the obligation of enterprises to draw up a Control Program Occupational Health Medical-PCMSO, and NR 9, creating the Program for Prevention of Environmental Risks-PPRE. The NR 17 of which addresses a more rational way the issue of ergonomics in work stations and NR 18 - established the Control Program and Work Environment - PCMAT, intended for construction, represented a major advance in the field preventions. Today's regulatory standards are revised from tripartite committees involving workers, technicians and entrepreneurs. Occupational diseases began to be better identified (MSDs work-related and noise-induced hearing loss, for example) and indemnity actions accumulate in the courts. Insurers are interested in the privatization of the insurance of occupational accidents

Even with a still hegemonic model of Occupational Health, under the control of capital, which conceals the extent of damage to workers' health and the environment, and supports herself with an institutional framework dichotomized, anachronistic and corporate (AUGUSTO, 1995), joint participation of workers and technicians in the area has occasioned hopes that despite the setbacks and losses highlighted throughout the struggles already cited by DAY (1994), the dream is made real by ensuring all workers decent health and safety expressing thus the quality of life and citizenship rights as claimed and not yet won. Health, as a universal right and duty of the state, is an achievement of the Brazilian citizen, expressed in the Constitution and regulated by the Organic Law of Health Under this law is the worker's health. Although the National Health System (SUS), in recent years has advanced greatly in ensuring citizen access to health care actions, only from 2003 national policy guidelines for the area began to be implemented.

These guidelines are:

- Comprehensive Health Care Workers;
 - Intra-and Inter-sectorial;
 - Structuring the Information Network on Occupational Health;
 - Support for the Development and Research;
 - Capacity Building of Human Resources;
 - Community Participation in Management of Shares in Occupational Health.
- Among the strategies for the realization of the Comprehensive Health Care Workers, we highlight the implementation of the National Comprehensive Health Care Workers (BRAZIL, 2005), whose objective is to integrate the network of SUS services and targeted

Tratado de Toxicologia Ocupacional

assistance surveillance, in addition to notification of diseases related to networking services sentinel (BRAZIL, 2004) 1.

Paracelsus - Swiss physician and alchemist (1493-1541)



"All substances are poisons, there is nothing that is not poison. Only the right dose differentiates a poison from a remedy." Paracelsus (1493-1541)



Bernardino Ramazzini, padre della Medicina del Lavoro

Bernardino Ramazzini (10/3/1633 to 5/11/1714) was an Italian doctor. Ramazzini was a forerunner in the use of a derivative of quinine to treat malaria. But his most important contribution to medicine was the work on occupational diseases called *The Morbis Artificum Diatriba* (Diseases of Labour) which related health risks caused by chemicals, dust, metals and other agents encountered by workers in 54 occupations. This was one of the pioneering works and the basis of occupational medicine, which played a key role in its development. He worked as a teacher dena University of Padua from 1700 until his death.

OATH OF AREA MEDICAL

Hippocrates, the Greek physician, 460-377th. C

INTEREST:

Consider also my teachers to my parents.

To teach this art, generously, to my and their children, considering them equal to my brothers, and those who have committed to pursue it, subject to this oath, and no other in contrary.

Apply the treatment to the benefit of patients according to my ability to consciousness, preventing them from any harm, even in order for anyone;

Never practice methods that cause abortion;

Preserve the dignity of my life and my art;

Log in private so only the patients for their benefit without corrupting the morals, nor do they cause harm or damage;

Secrecy of whatever I see, hear or come to know the practice of medicine or elsewhere, should not be disclosed, given the discretion as a duty;

If I fulfill this oath and violating in any way, let me enjoy my life and my art, enjoying eternity, fame and honor among men.

If I transgress and perjure is otherwise my fate.

I swear and promise that the practice of medicine, will always be faithful to the demands of honor, the principles of science and duties.

Give my free professional care to the poor and not require my ever higher salaries to my work.

Admitted to the intimacy of home, my eyes do not see what goes on there, my language will be changed to know the secrets entrusted to me, and my profession does not serve to corrupt the morals, nor to encourage the crime, nor will practice methods causing abortion.

I will deal with the same equal footing, rich and poor.

SANTA BECOME MY LIFE AND MY ART. I promise to keep this oath to the extreme limits of my strength and my life. If this does not

Suelen Queiroz

meet my commitment, that is my fame and my destination negligible compared to men.

HISTORY AND CONCEPTS OF TOXICOLOGY

In ancient China, there are reports of Emperor Shen Nung, who lived around 5000 a. C. (Also known as Emperor Yan, "the divine farmer" for having introduced the advent of agriculture in ancient China, and also as the father of Chinese medicine since tested about 365 herbs and possibly died due to toxic doses thereof) and was responsible for composing a treatise on herbs that in turn was being updated by future generations, which explains the deep knowledge of Chinese people with regard to medicinal herbs. Following the saga of toxicological knowledge, appear around 1500 a. C., the Ebers Papyrus, one of the oldest preserved documents with toxicological information to the present day. In 1862, in Luxor, the papyrus was purchased by Edwin Smith, American adventurer who lived in the city of Cairo, and remained with the paper until 1869, when he put up for sale. In 1872 the papyrus was purchased by the Egyptologist Georg Moritz Ebers novelist, hence the name coming Ebers Papyrus. Papyrus is a comprehensive report of the medical history of ancient Egypt. In it are given as knowledge of the human body, structure and cardiac and vascular healing substances prescriptions for various ailments caused by toxic agents of animal origin when both vegetable and mineral. After 100 years, in Sumer (1400 BC), texts related to a mythological figure called Gula, were associated with charms, spells and poisoning, showing that the toxicology, again, as elsewhere, was linked with magic and power.

In line with developments, Homer describes the use of poison on arrows in their world-famous works "The Odyssey" and "The Iliad." Socrates is sentenced to die by drinking hemlock bitter (with great power plant alkaloid toxic) and his nemesis. After 100 years, in Sumer (1400 BC), texts related to a mythological figure called Gula, were associated with encantos, spells and poisoning, showing that the toxicology, again, as elsewhere, it is on core and power . In line with developments, Homer describes the use of poison on arrows in their world-famous works "The Odyssey" and "The Iliad." Socrates is sentenced to die by drinking hemlock bitter (with great power plant alkaloid toxic) and his nemesis already demonstrating a good understanding of the mechanism of toxic action, the famous Greek philosopher said that after sucking the liquid remains no longer feel up to walking legs because thereby accelerate the effect of the drug. Also Alexander the Great, one of the great conquerors of antiquity and the king of Macedon (a pupil of Aristotle, Plato's pupil, a disciple of Socrates) died from poisoning.

Numerous other historical figures such as legendary Mithridates IV, Cleopatra, the components of the Borgia family, Leonardo da Vinci, Paracelsus, Catherine de Medici, J. Mateu B. Orfila has compulsory service in any text on the history of Toxicology, represented by the importance in the evolution of this science, each with its own unique contribution.

When Paracelsus postulated that the difference between healing and poisoning is the dose, established some theoretical toxicology as a scientific discipline, where there was a subsequent universal convergence. With that, was a new conceptual domain, breaking with common sense, leaving aside the "magic potions" people of his time. It was a split within the then-current empirical knowledge of civilization. But it was only in the nineteenth century that toxicology was configured as modern science, and within all the transformations that gave overwhelming your life and think of Europe at that time, especially with the ways that the development of chemistry was opening.

Toxicology not limited to the findings of toxic effects. He also sought to discover and understand the mechanisms of action of toxic substances. No longer just a descriptive and analytical science and has acquired an experimental nature (Moraes, 1991). Thus, absorbed a recent approach that is preventing, through its application for recognition, identification and quantification of risks.

Brazil

Obviously the question toxicológicaé also known in Brazil for several centuries. Since the Indians, who have exhibited this type of information until the black slave, who brought their ancestral knowledge of relevant practices and religions of their land, and Portuguese, with the knowledge of the European season. From the standpoint of Toxicology configured here as a matter of study, research and teaching, we can say that it emerges strongly in the year 1950. In 1976 takes place in the city of Manaus, the name, the first technical event - scientific toxicology registered in Brazil.

In 1977, in the seaside city of Guarujá, Sao Paulo, is the First Brazilian Congress of Toxicology, which discussions remain only in memory of the brave survivors who have it participated.

After 15 successive editions of congress, since that first in Guarujá, Brazil toxicology grew, and today is represented by a broad, multidisciplinary and multi-family toxicologists. House mater of our toxicology, the Society of Toxicology (SBTox, [www. Sbtox. Com. Br](http://www.Sbtox.Com.Br)) has organized several conferences and all these other events, fulfilling its

role to disseminate and assert that science among us, prompting the Brazilian population and the authority of the country for the most important facts related to it. Among others, we must also emphasize here the existence and cooperation of institutions and organizations such as the Brazilian Society of Ecotoxicologiae Analyst Group of Pesticide Residue.

CONCEPT OF TOXICOLOGY

It's part of physiology that studies the body's response to pathogenic chemical agents.

FOOD, MEDICINE, POISON

All substances are poisons; there is nothing that is not poison.

Only the right dose differentiates a poison from a remedy. "Paracelsus (1493-1541). Fonz-Diacon, in its toxicology, offers the following definition:" poison "is a defined chemical substance, introduced into the body, acts, until the toxic dose in proportion to the mass and cause disorders, which may cause death. "Sydney Smith said that" it is almost impossible to define the word poison, because certain substances hazardous to life. in certain circumstances, prove to be innocuous in others, and how many things can be absorbed, arrive to take place hazardous to health, taken in excess "

DIVISION OF TOXICOLOGY

Paying attention to the studies of the toxicology of harmful substances is divided into:

- I-prophylactic
- II-Industrial
- III clinical
- IV-Forensics
- V-analytical

I-TOXICOLOGY PROPHYLACTIC

Nocivous study of the chemical environment in general. Identifies toxic, its origins and quantities. Propose precise and hygienic measures necessary to control health

Avoiding large-scale poisoning of the population. Prophylactic Toxicology intended to maintain the safety limits for humans exposed to the action of several chemical agents dispersed in the environment.

II-INDUSTRIAL TOXICOLOGY

Aspects are considered:

- a) Chemicals: producing new products that are intended for therapy, medication, should be isolated and synthesized;
- b) Hygienic, for the middle environment for insecticidal, fungicidal, esthetic as cosmetics, the comforts of home (paints, bleaches,

detergents, adhesives, etc.);

c) Surgical, as antiseptics, etc. Example of the latter can be remembered in relation to the pharmaceutical industry, following the dramatic effects of thalidomide were by regaining their care.

III-CLINICAL TOXICOLOGY

Through the study of symptoms and clinical signs that harmful effects of chemicals produced in the human body to diagnose clinical toxicology demand and guide appropriate therapy. It is recommended, always, that any form of intoxication or poisoning has special clinical attention, even in patients with severe intoxication that was not followed by death.

The parallel is always present in the light of industrial economic race: the launch of new chemicals in the pharmaceutical market new toxic cause.

The iatrogenic poisoning.

IV-FORENSIC TOXICOLOGY

It is significant value to the satisfaction of justice, either by clinical workup, such as by chemical analysis, it is able to identify the poisons of any species and guide the dictates persecuted. The importance of studying Forensic Toxicology from the need to establish, in cases of death by poisoning, if it were from suicide, accident or crime. The forensic toxicology has expanded in recent years as its field of action, which is practically freed of legal medicine, constituting part of the discipline. Programs in Forensic Toxicological matter is studied very superficially, aiming only to provide some more knowledge needed by coroners and lawyers.

CHAPTER 1

Poisoning

Poisoning is the act or effect of intoxicants. Poison is poison. It is the introduction of toxic substance in the body. Depending on the type of toxicology expertise can be considered in several ways. Interest in the present case the professionals, since the object of occupational medicine. Can be matched in special circumstances food caused not only by toxic foods, plants or animals, for chemical additives for the narcotics and drugs and by drugs.

The formal diagnosis in intoxicated person must meet the following: a) history, b) clinical examination, c) laboratory tests and clinical toxicologic necessities. The physical examination (clinical) should be thorough and very careful attention to the verification of the pupils, of fever, blood pressure, pulse, heart rate, skin, oral abnormalities, neurological disorders, etc. The clinical history should raise with details of family members or third parties, and events that occurred before involving the patient with intoxication. In cases of occupational intoxication due to stress the importance of detailing the activities carried out and highlight the possibility of continuous exposure and intermittent or permanent employment to the chemical contaminant. Are frequent symptoms of some types of poisoning:

a) Ocular changes: mydriasis (dilated pupils), atropinic poisoning by belladonna plant, solvents, tobacco, depressants, etc.), miosis (pupils contracted), (organophosphate poisoning, opiate

b) anomalies mouth: dry mouth (poisoning atropine, belladonna plant, etc.), hypersalivation (pesticides, heavy metals), gingival (heavy metals);

c) neurological disorders: a coma (poisoning by alcohol, barbiturates, opiates, tranquilizers, salicylates, organic solvents, cyanide, antihistamines), seizures (organochlorine pesticides, organophosphates, strychnine, aminophylline, etc.), tremors and muscle jerking (insecticides organophosphates, nicotine), paralysis (heavy metal poisoning), headaches (carbon monoxide poisoning, nitrite, nitrate), d) urinary disorders: Urinary retention (atropinic poisoning), hematuria (anticoagulants), anuria (toxic disturbances that determine .

PENETRATION IN THE BODY

Penetration (from the Latin "penetration") lies in the act of

penetrating ouefeito.

The velocity and absorption depend on the route of penetration. but also the effects, neutralization and elimination of possible toxic tóxico.Os (or poisons) and the drugs can be delivered orally, gastric, rectal, inhalation, dermal, subcutaneous, intramuscular, intraperitoneal, intravenous, intraarterial, intraosseous and intrathecal . The most common are the oral and gastrointestinal. The drug can be absorbed at the oral mucosa or swallowed, the level of gastric and intestinal mucosa. So you can act the poison needs to reach the arterial system and its effects will manifest capilar.Aí. The hair system is the field of action of poisons (Fonz-Deacon), so that quick açãodo poison depends on the path more or less that has to do to reach the capillary system, as well as the obstacles in its path.

TOXICITY

Is the ability of chemical agent have harmful effects to the body humano.E always measured in relation to other agents and varies among species and within the same biological espécie.O coefficient (toxicity) is the toxic dose per kilogram dose Animal.as is considered fatal one that can eliminate 100% of the same species, the average fatal dose is one that is able to kill 50% of those animals.

It is produced by the toxic effects:

a) desired exacerbated, as bleeding consequent to the use of anticoagulants.

b) a number of points as bronchial asthma consequences of the use of salicylates lethal (cardiac arrest consequent to the use of anesthetic).

The lower the average fatal dose is considered the more toxic the penetrating agent. For human toxicity is rated:

- a) extremely toxic.1mg/Kg Or less;
- b) highly toxic:. 10 to 50 mg / kg
- c) moderately toxic:. 50 to 100 mg / kg
- d) slightly toxic:. 0, 5th 5 g / kg

Poison ENDEMIC

Are those produced by natural means in the presence of toxic environment. The main ones are: a) chronic regional endemic hidroarsenismo, which occurs in regions of Córdoba, San Luis, Santiago del Estero;

b) hidrofluorose that occurs in the south of Buenos Aires and the pampas.

SOCIAL Poisoning

Are those that have profound social consequences for the inconvenience psicomorais people. The in producing what can be said of addiction and poisoning anticoncepcionais. Elas cause euphoria, tolerance, habit, symptoms of dependence and withdrawal symptoms and can be considered toxicomanígena.

The euphoria estadofalso consist of well-being with demonstrations hilariantes. O habit in need of a progressive increase in doses to achieve the same effect (some even lethal doses). Tolerance reduces the capacity developed the habit of tolerating doses considered noxious or psychic dependence letais. A is an uncontrollable desire claiming droga. A physical dependence is a state that is characterized by the appearance of physical symptoms when the drug is discontinued. Abstinence is summarized in the syndrome characterized by a set of symptoms and signs opposite to those produced by the drug.

GENETIC Poisoning

Are those relating to enzyme changes were transmitted by inheritance or idiosyncratic. The action of a chemical on the organism is followed by metabolism or by storage. The termination of the effect occurs with the complete elimination. The metabolic result can give a product in more or less toxic.

Thus, when an enzyme catalyzes a reaction that makes a very active chemical agent other less active, lack of this enzyme causes the toxicity of the chemical agent is greater and more prolonged. It is known that genetic characteristics are transmitted through the genes I study these problems is to affect pharmacogenetics.

Poison Iatrogeny

Paradoxically are those which arise as a result of drug use, the higher dose, summation, synergism, or else by hypersensitivity error indication, or the method of application, giving rise to the emergence of the desired effects and exacerbation of colaterais. A responsibility is assigned doctors, paramedics and the communities affected persons and they do not consider the recommendations therapies. Also included are those prescribed by traditional healers, or infusions, poultices, enemas, prepared by the lay person or bystanders, but for therapeutic purposes.

Can (iatrogenic poisoning) produce:

- a) defects (thalidomide, selenium)
- b) neuropsychiatric disturbances (seizures and parkinsonism; corticosteroids neuritis, arsenic and antimony; Kernícteros; vitamin K in

newborn infants, encephalopathy)

d) metabolic disorders (gout secondary, mustard, radioactive phosphorus, TEM-tri-ethylene-methenamine, demineralization, cortisone long);

Grow day by day the numbers of iatrogenic poisoning, shock allergic to penicillin or certain anesthetics. There is no doubt that the pharmaceutical industry today presents new "weapons" therapeutic elementary values, which were responsible for the elimination of many diseases, particularly infectious, but those same "weapons" are thrown in the trade without needed care and increase the "therapeutic risk" by the lack of oversight in sales.

Food poisoning

Are those that occur by ingestion of contaminated food. Contamination can take place by:

a) chemicals, metals, pesticides, preservatives, artificial coloring, antioxidant of grease, oil etc..

b) chemicals in the food itself: fungi, mollusks, fish, peas, cassava,

c) bacteria: staphylococcus, salmonella, proteus, enterococci, clostridium botulinum (due to presentade disease germ itself and its toxins, which is why it is called food poisoning of toxins. Such bacteria are excellent culture media in the jerked beef, bacon, No ham, sausage on in sausages, in salad, mayonnaise, eggs, cream, candies and ice creams. The fish, shrimp, oysters, mussels have rapid decomposition that promote and facilitate bacterial growth.

Receive the special designation mycetes intoxication by fungos.são most venomous species: Amanita phalloides, verna and Vitrolick; amanita muscarinic and panthere, glyromitaesculento. It features food poisoning in a short period of time between food and symptoms, so that the presence of the pathogen in the body thus favors.

DEFENCE ORGANISATION

The path of the poison is not done freely, without obstacles. The body tends to defend itself, the extent of their forces. The first obstacle is that the poison is in the viscera figado.Esta enjoys the property of their tissues fixed in certain foreign elements in the form of organic compounds.

Minimal doses of lead may well be arrested, after being eliminated by bile. Only large doses of lead can cause death rapidamnete, crossing

the barrier liver. The liver can also modify chemical Anatur certain alkaloids and mitigate them toxidez.Os bones also cooperate in the defense orgânica.O arsenic, mercury salts, absorbed in large doses, can reach the capillary system and determine the death in small doses

The body's defense in explaining why certain poisons, highly toxic when absorbed by the blood directly, bypassing the liver, do not exert any harmful effects when they enter via the gastrointestinal tract.

The snake's venom, for example, deadly when it enters directly into the circulation after the bite, can be absorbed by sucking the wound, without any danger.

The lungs also act defensively in favor of a gas economia.Quando is ingested into dissolution, before reaching the capillary system, it must go through pulmões.Ora, effecting the lungs, gas exchange between the organism and the environment: gas exhaled carbon dioxide and oxygen is fixed, the venous blood, dark, it is arterial blood, red radiance.

The lungs, eliminating, at least in part the toxic, gaseous, volatile, such as carbon monoxide, hydrogen sulphide, chloroform, alcohol, ether.

The leukocytes also take part in organic defense (phagocytic action).

BESREDKA demonstrated that when introducing under the skin of an animal, a small amount of a compound soluble or insoluble arsenic (arsenious anhydride, trissulfureto arsenic), it produces a hypercellularity, which maketh the body of these toxic through a variety of fagocitiose.O arsenic mineral is disaggregated and transformed into organic arsenic, much less toxic, which is later eliminated by the kidneys.

Finally, the body defends itself by vomiting and diarréia.Em general, toxic substances have strong emetic properties, so that most of the poison can be rejected by vomiting before absorçãotenha been sufficient to cause death. Evacuations Alvin befallen the ingestion of certain toxins, expel the body's still a part of the venom.

Thefactors INFLUENCE ON THE ACTION OF POISONS

The division of a state of toxic influence favorably on his absorçã.O arsenious anhydride, for example, administered in powder form, will act less quickly than in solution because the first case the poison will be absorbed only in proportion to their slow dissolution in body fluids, while the second hypothesis is immediately absorbed.

Foods that are eaten while the toxic substance, can facilitate or retard their bodies absorçã.Os acids facilitate the absorption of phosphorus by dissolving it and allowing it to penetrate the bloodstream, rather it will slow the absorption of a poison also fearful, arsenious

anhydride.

The state of emptiness or fullness of the stomach also has an influence on the uptake of venenos. Se the stomach is empty at the time of ingestion of toxic, although not diluted in the mass food will soon absorvido. No otherwise it will be slower absorbed and more easily found in the liver or rejected by the bile and urine.

However, its action will cianureto de potassium maximum in the case of repletion of the stomach is that the potassium cyanide is toxic, giving off hydrogen cyanide under the influence of hydrochloric acid in gastric juice that is secreted when the stomach contains food.

The body weakened by the disease less resistant to the action of poisons in a state of saúde. Certas people are more sensitive to even a poison than others, this is called susceptibility particular idiosyncrasy.

Finally, the age also influences on atividadedos poisons: a child succumbs to the ingestion of a few drops of laudanum, while an adult may absorb several ounces without drawbacks should also be sérios. O costume assinalado. Permite body to resist the action of a massive dose that would this is deadly without treinamento. Chama to that fact Mithridates, Mithridates word derived from the king of antiquity, who, fearful of being poisoned, became accustomed the action of poisons.

MODES OF ACTION. EXPLANATORY THEORIES

The toxic substances act on the body very variable in its intensity, speed and mode ação. Alguns poisons kill quickly acting on major organ systems, blood system, nervous muscular. Outros act slowly, serving many vezes sobre the glandular system .

REMOVAL OF POISONS

In what way are eliminated poisons in our body? Some pass through the body and are eliminated without any change in its chemical makeup. Most alkaloids (strychnine, morphine, brucine, nicotine) are rejected by the urine, though nothing has changed in their molecular structure (in the poisoning, the alkaloids being always absorbed in excess, not only that this excess is not modified in the urine).

The carbon monoxide, hydrogen cyanide, chloroform, ether etc.. Are still in the same case. Other bodies, in contrast, are modified during their passage through the body and form rejeitados sob diferente. Alguns are reduced, the bromates bromureto pass to the state of the state of the iodates iodureto, but more often than not, they are oxidized to expense of oxygen sangue. Os sulphides, sulphites, thiosulphate become sulfates etc. extensions. Some times the toxic nature of change by a double

decomposition, which occurs in the stomach between the elements of gastric juice and veneno. Assim, small amounts of lead acetate give rise to insoluble lead chloride, the contact of acid gastric juice cloridricodo, and acetic acid inofensivo. Tenham sofridomodificação or not, drugs are eliminated more or less slowly in the body, which always tends to get rid of foreign elements .

ROUTE OF ELIMINATION

The routes of elimination of toxic substances, or products resulting therefrom, are the devices of secretion or excretion, such as the liver (the bile), kidneys (in urine), the various glands of the economy (sweat, saliva, mucous membranes the skin) and finally to the gaseous substances (carbon monoxide, hydrogen sulfide, alcohol, etc.), the lungs.

But the substances eliminated by the salivary glands are partially reabsorbed with the bolus; eliminated by the bile partly pass faeces, but can be reabsorbed in the intestine fortunate that the great route of elimination is the kidney.

Only the study of each particular venenoé allowing explicit reference to the duration of your elimination period.

SPECIES DEVENENOS

The three kingdoms of nature can provide toxic substances so that we have mineral poisons (Copper sulphate, lead acetate), vegetable poisons (strychnine, morphine), which are more formidable poisons animals, discovered the end of last century, almost simultaneously, the Italian and French SELMA ARMAND GAUTIER (leucomáinas and ptomaine) . The leucomáinas organs are produced during pathological conditions, and ptomaine stem from rotting organic matter.

GASES OF ROOMS:

The carbon monoxide is odorless substance, colorless, density equal to 0.96, with a blue flame burning, and very toxic to the human body. Their usual sources are fumes from burning coal, the braziers, fires and explosions. Between us is almost always as the original source of the gas street. The common gas that we use in kitchens, is from the calcination of coal and presents a complex composition in which we can distinguish hydrogen, methane, carbon monoxide, nitrogen, acetylene.

The carbon monoxide poisoning as superagudaseriam reported cases of sudden and massive inhalation. The victim is only three to four breaths and is immediately attacked the tremors, dizziness, loses consciousness and dies. The common thread, however, is the acute form,

in which the patient has headache, hallucinations, tinnitus, scintillating scotoma, impotence, muscle, nausea, vomiting, convulsions and coma. O can last hours or days, and the more fatal will be the prognosis the longer its duration. If the individual is restored, has many times, confusional state, delirium, neuritis, paralysis, hyperesthesia, neuralgia, xanthopsia, blindness, tinnitus, dizziness, edema and trophic skin disturbances, aspiration pneumonia, etc..

Besides the two preceding forms, there is still a chronic form of poisoning oxicarbonada, usually professional (cooks, firemen, etc.). The patient complains **psychic disturbances** with intellectual torpor, loss of desire, insomnia, etc.; **motor** as asthenia; sensitive as rheumatic pain, epistaxis, nausea, vomiting.

Poison Iatrogeny

Paradoxically are those which arise as a result of drug use, the higher dose, summation, synergism, or else by hypersensitivity error indication, or the method of application, giving rise to the onset of exacerbation of the desired effects and side effects. The responsibility is assigned to doctors, paramedics and affected people themselves and they do not consider treatment recommendations. Also included are those prescribed by traditional healers, or infusions, poultices, enemas, prepared by the lay person or bystanders, but for therapeutic purposes.

Can (iatrogenic poisoning) produce:

- a) defects (thalidomide, selenium)
- b) neuropsychiatric disturbances (seizures and parkinsonism; corticosteroids neuritis, arsenic and antimony; Kernícteros; vitamin K in newborn infants, encephalopathy)

ENVIRONMENTAL Poisoning

Are those that occur in the environment (air, water, soil). In big cities is that most occur, since in these focus industries, the cars polluting and contaminating the etc. All ambiente. Veja the case of Cubatao, considered the most polluted city in the country

Losses are known organisms that cause air pollution to man; a) death, b) chronic diseases (bronchitis, emphysema, asthma). c) recurrent episodes of morbidity (decrease in vital capacity), d) minor disorders and functional reduction (discomfort, respiratory infections);

e) psychological effects (reduced visibility, odor, irritation, trachea and nose). The pollution by sulfur dioxide, one of the most common pollutant, produces the following effects;

a) The effects of concentration are: I-health effects, especially increased risk of diseases affecting the respiratory system; II-corrosion surfaces metálicase their effects on certain types of stone and painted surfaces; III-effects on life plants because of the influence on photosynthesis and other vital.

b) Effects of acid deposition because of the rain water and they are: I-effect on lakes and rios. II and effect on soil

Occupational poisoning

Are those of the person from exposure to chemicals in the exercise of their activities. These are the areas that matter most to infortunística.

Are presented for acute onset and crônicas. Têm increased nowadays due to the rising number of chemical agents and / or toxic that a worker has contact.

Intoxication may be defined as professional occupational disease or work (and the acute recognition as an accident within the meaning of the term by some medical experts).

The occupational medicine or occupational has devoted more attention especially after the "package" of March 1986 Decree-Law No: 2284 1986, by recent numbers of new jobs in industries and commerce in general. The pulmonary diseases are the most common chronic and acute poisoning today. May be related:

a) mineral pneumoconiosis (caused by silicates) (kaolin, talc, mica, and asbestos), for coal, for iron, graphite, tin (estano) barium sulphate (baritose) for aluminum, beryllium by, for nickel; cadmium; by vanadium by selenium, tellurium by tungsten

b) pneumoconiosis caused by organic cotton, sisal, jute, bagasse, etc..

c) occupational diseases caused by soluble gases and aerosols, ammonia, chlorine, nitrous oxide (fertilizer), etc..

d) Special mention needs to pesticides, smoking, rodents, herbicides, insecticides, fungicides, repellents, since they provoke often victims both in handling the preparation, the work itself, as in spraying and / or use.

Cancer is produced by professional:

a) tar, tars and oils, arsenic, x-rays (the skin);

b) benzidine, aniline, diphenylamine, auramine, xenilamina, betanaftamina (bladder);

c) benzene, radiation (leukemia)

f) radioactive substances (bone cancer)

DISEASE OF WORK:

a) Any so-called occupational diseases inherent in certain industries and related act in the Ministry of Labour.

The degenerative disease is not inherent in either age group, resulting from special conditions or exceptional in that work executed, provided, directly related to the activities performed, causing reduced capacity for work that would justify the granting of disability benefits under the Act.

OCCUPATIONAL TUMORS

Tumors may arise from occupational exposure to the carcinogenic action or result of trauma in the etiology of skin coat tumors, one should not disregard the existence of conominantes causes, such as individual constitution, lifestyle habits, especially as regards to chronic poisoning, such as alcoholism, the maintenance of poor nutrition and rest.

CARCINOGENIC CHEMICAL AGENTS

Exposure: Arsenic, Chromium, Coal, aromatic amines, nickel, oil and heavy metals.

CHAPTER 2

OCCUPATIONAL DISEASES

Data from 1991 estimated at 100,000 the number of mining assets and reported about 400 000 workers involved in mining activities. In the manufacturing industry to the IBGE, in 1996, estimated at 8, 5 million workers in activity, with about 43% of potentially exposed to dust. In the same year, the estimate was in the construction of 4, 5 million workers. The agricultural sector, in turn, had 16, 7 million workers exposed to organic dust mainly. Recent data for estimation of exposure to silica in Brazil indicate that for the period 1999 to 2000, about 1,815,953 workers with formal jobs were exposed to silica for more than 30% of your journey involves exposure to asbestos work. about 20 000 workers employed in mineral extraction and processing (mining of asbestos, asbestos-cement products, friction materials, specialty papers, seals and gaskets and textile products). This number dropped in the last four years due to market loss and replacement of asbestos in some industrial products. However, it is estimated that another 250 thousand - 300 thousand workers are exposed inadvertently in the sectors of construction and mechanical maintenance. The extraction of coal currently employs (2004-2005) three thousand - four thousand miners. It is an activity that has floating numbers, depending on energy policy, demand and price of coal.

Epidemiological data from several countries show that the risk of pneumoconiosis is still a problem worldwide both in developed countries as in developing, although in the working conditions and job insecurity and environmental control of individual exposure, lead to a greater risk.

In the 90s, for example, have reported outbreaks (clusters) of silicosis in countries like France, Italy, Netherlands, USA, Canada and Finland, although mortality from silicosis in these countries have fallen dramatically in recent decades. In countries like South Africa, in the same decade, the estimated incidence of silicosis among miners was 20 to 30%. At this high rate is associated with the high risk of tuberculosis and the high prevalence of infection HIV. Da Similarly, researchers in countries like China, India and Brazil have published results from studies with high prevalence of silicosis, showing the existence of the problem and the need for improvement in diagnosis and control exposição. Os epidemiological data on pneumoconiosis in Brazil are scarce and refer to some of these areas of activities in situations focais. Os data are available about the

occurrence of silicosis, for example give a partial idea of the risk related to this pneumoconiose. A larger national sample of silicosis is in the mining of underground gold of Minas Gerais, where there have been about four thousand cases. Other studies have investigated important in the ceramic industry. Some cross-sectional descriptive studies published so far give a frequency of silicosis ranging from 3, 5% in the quarrying sector (exploration and production of granite gravel) to 23, 6% in the sector of shipbuilding operations (blasting sand). With regard to exposure to asbestos or asbestos, the few published studies show a prevalence of 5, 8% of asbestosis in asbestos-cement industry (manufacturing of roofing and water tanks) and the occurrence of 74 cases of asbestosis (8, 9%) , and 246 cases of pleural plaques (29, 7%) in selected population of former workers of that sector of the asbestos industry. In coal mining in Brazil, restricted to the South, there are over 2,000 diagnosed cases of PTC. The point prevalence of PTC in mining assets in the 80's was 5, and 6% probability of occurrence was estimated at 20% after 15 years working underground. Series of clinical cases and other cases of pneumoconiosis have been described over the years, warning of possible occurrence of diseases related to exposure and iron oxide, phosphate rock, talc, abrasives, hard metals, beryllium and sericite.

1.4 PATHOGENESIS AND PATHOPHYSIOLOGY

For the occurrence of pneumoconiosis is necessary that the particulate material to be inhaled and reach the lower airways, in concentrations high enough to overcome the clearance mechanisms: the mucociliary transport, lymphatic transport (known as clearance) and phagocytosis by alveolar macrophages. Mucociliary transport is predominantly provided by the mucociliary system up (80%) through the ciliary system from the terminal bronchioles.

About 20% of the pulmonary transport is carried by the lymphatic system, which receives or free particles phagocytosed by alveolar macrophages. Pneumoconiosis is a disease by inhaling dust, substances that the body can fight with his little immune defense mechanisms and / or leukocytes, unlike what happens with microorganisms that can be phagocytosed, digested or destroyed by the action of antibodies and immune cells through lysosomal enzymes and other mechanisms. To be effective in reaching the lower respiratory tract the particles should have a median aerodynamic diameter less than 10 μ m, because above this size are retained in the upper airways. The respirable fraction (<5 μ m) are more likely to be deposited in the lower respiratory tract (respiratory and terminal bronchioles and alveoli), and initiate the inflammatory process that is perpetuated by the chronic inhalation and / or quantity that exceeds

the defenses, may lead to lung changes. Particles with diameters of 5 to 10 μ m, although to a lesser extent, also afford to be deposited in these regions and produce disease.

The pulmonary reactions to inorganic dust deposition in the lung will depend on physicochemical characteristics of the aerosol (eg, smaller particles and freshly fractured silica fibers thinner and longer in the case of asbestos, are more harmful) dose (which depends, among others, concentration in inhaled air, the volume per minute and duration of exposure), presence of other dusts, previous pulmonary disease, which can be modulated by immunologic factors individual and in many cases by smoking.

Non-fibrogenic pneumoconioses:

characterized, the histopathologic point of view, by type of macular lesion interstitial peribronchiolar deposition of particles phagocytosed or not, with no or slight degree of structural disorder, and mild inflammatory infiltrate around, with none or mild fibroblast proliferation and fibrosis. Dependent on the knowledge of the kind of dust inhaled, pneumoconiosis and siderosis leads specific name (Fe), baritose (Ba), estanose (Sn), in order etc. Tendo histopathological pattern formation and deposition of stains isolated, without producing fibrosis, respiratory dysfunction is virtually absent and the clinical course is considered benign when compared to the possible evolution of fibrogenic pneumoconioses. Despite the absence of fibrosis, the radiation pattern of change is quite similar to silicosis, micronodular or reticulonodular opacities with diffuse. The radiopacity is due to the presence of metal / mineral deposits in the interstitium

In some cases, as in baritose and in some rare cases of siderosis, the possibility of regression of the radiological picture from the elimination of deposits through the clearance macrophage-linfático. Em other cases, however, depending on the dose of inhalation duration of exposure is prolonged and the particles are very fine (<1 μ m) deposited dust can cause a tissue reaction with production of various degrees of fibrosis. **Fibrogenic pneumoconioses:** As the term says are the pulmonary reactions to inhalation of particulate matter that leads to interstitial fibrosis of the lung parenchyma. The following are briefly discussed the mechanisms involved in the development of major diseases covered in this manual.

•**Silicosis and asbestosis:** the processes of installation and development of silicosis and asbestosis are similar, although the first results in a focal interstitial fibrosis, which begins with the formation of

granulomas concentric deposition of collagen, and the second with the proliferation of collagen in the interstitium, without the presence of inflammatory cells relevant to defense, which was not well understood yet. Inhaled particles (silica or asbestos) in contact with water and within alveolar macrophages after being engulfed induce the formation of reactive oxygen species (ROS) and nitrogen (RNS) which stimulate (through the activation of nuclear transcription factors) cytokine production by macrophages, responsible for attracting to the region alveolar inflammatory cells (lymphocytes, mast cells, neutrophils), which in turn release more cytokines and ROS and RNA. This process eventually induce an alveolitis with lesion of type I pneumocytes, proliferation of type II pneumocytes and fibroblasts, passage of particles into the interstitium and stimulating the proliferation of interstitial fibroblasts to initiating fibrogenesis. If inhalation of particles has been great, or persist in time, the inflammatory process with cellular damage, proliferation, apoptosis, and fibrogenesis, there is still installing the diffuse and progressive fibrosis of the lung parenchyma.

•Hypersensitivity pneumonitis:

is characterized by acute episodes of mononuclear cell infiltration and accumulation of fluid in the airspaces and interstitium, a few hours after contact with antigen. In this process, macrophages play an important role, in contact with inhaled antigen, release cytokines that attract inflammatory cells giving rise to inflammation, which may manifest clinically with fever, cough, dyspnea, headache, myalgia, these symptoms were of short duration (1 to 3 days) and that resolve spontaneously. Repeated exposure to antigen, leading to recurrent exudative pneumonia, may evolve into a chronic form of the disease with non-necrotizing granuloma, bronchiolitis obliterans and diffuse interstitial fibrosis. The mechanisms involved in its phase of activity usually responds to corticosteroid treatment.

•Hard metal pneumoconiosis:

the mechanism of disease involves an inflammatory reaction triggered by the alloy, which is manifested by cellular and humoral immune mechanisms, presenting pictures of subacute alveolitis or insidiously progressing to interstitial fibrosis with the presence of bizarre giant cells occupying the alveolar spaces and interstitium, coexisting phases of desquamative interstitial pneumonia and chronic fibrosis.

•Coal workers' pneumoconiosis (PTC):

workers' pneumoconiosis in coal dust deposition triggers an

inflammatory process initially orchestrated by alveolar macrophages, with lower intensity than those generated by silica particles, but sufficient to cause injury to the alveolar epithelium. As a result, until the passage of particles into the interstitium and begins the formation of accumulations of coal and macrophages with engulfed particles, around the respiratory bronchioles, with the presence of reticulin fibers and deposition of small amounts of collagen.

These injuries, known as coal macule, measuring about 1 to 6mm. Intralobular are little or not visible on radiographs and usually accompanied by focal emphysema adjacent to areas of the macula. With progression of the disease, resulting from inhalation or continued even after the cessation of exposure, may develop in larger nodules of about 7 to 20mm, with presence of macrophages with pigment inside them, presence of reticulin and increased amounts collagen. With chronic exposure, the nodules may coalesce giving rise to the form of progressive massive fibrosis (PMF). The PMF is usually bilateral, predominantly upper lobe, middle lobe and superior segments of lower lobes. They are usually asymmetrical, sometimes showing characteristics of malignant tumor, which may cavitate, with the patient expectorating blackened material, known as melanoptise. The lesions are distinguished by the FMP by silica present, histologically, higher reticulin / collagen, large amount of coal dust, dense arrays of reticulin and collagen and absence of silicotic nodules. The FMP evolves more frequently with dyspnea, mixed respiratory disorder, pulmonary hypertension and cor pulmonale.

•Berylliosis or chronic beryllium disease:

is a granulomatous pulmonary disease, whose evolution can lead to chronic interstitial fibrosis of lung parenchyma, resulting

of immune response to beryllium inhalation. Presents three important features: 1) can be triggered by low doses or short exposure (<1 year) 2) manifests itself after a long latency period (usually> 10 years after start of exposure) even though the

individual away from exposure for several years, and 3) less than 5% of exposed individuals develop the disease, probably due to greater genetic susceptibility. It is indistinguishable

of sarcoidosis and, unlike other pneumoconiosis, can be treated with corticosteroids.

•Mixed dust:

mineral dust aerosols are mixed with low silica content, such as mica, sericite, kaolin and others. They can produce pictures of nodular fibrosis, different classical silicosis. In some recent studies, these nodules proved to be starring on histopathology (known as "Medusa head injury"), with progression to diffuse fibrosis from them, giving a radiological reticulo-nodular. Occasionally, they occur with pneumoconiosis PMF. Importantly, the type of change might not be homogeneous parenchymal full extent of the lung. It is not uncommon prevalence of pneumoconiosis nodules (eg, nodules or silicotic nodules in mixed dust) in the upper lobes and the prevalence of interstitial fibrosis at the bases of the same lung in cases of exposure to dust of silica with restricted content. The histological diagnosis will depend on the predominant changes found that, in short, is a reflection of the area of lung sampled. In coal may also occur in rheumatoid pneumoconiosis or Caplan's syndrome. Affected patients have circulating rheumatoid factor and pulmonary nodules with central eosinophilic, granular and necrotic, with fragments of collagen, elastin calcification and sometimes cavitation. Exposure to silica is also associated with an increased incidence of other autoimmune diseases such as scleroderma and renal glomerular disease. Pneumoconiosis is studied within the chapter of interstitial lung disease. The common characteristic of this group of diseases is the functional constraint for interstitial fibrosis and the consequent reduction of the expansion of the parenchyma, associated with barriers to gas exchange. In general, there was constraint functional only in cases advanced pneumoconiosis.

The decrease in gas exchange occurs earlier in pneumoconiosis that cause diffuse fibrosis, since the nodular fibrosis (eg, silicosis) preserves areas of normal parenchyma between the nodules, sufficient to

maintain a normal function of gas exchange, until the later stages of the disease, when much of parenchyma

fibróticas. Areas are replaced by deposition of mineral dust in large and small airways, can generate tables of chronic bronchitis, for direct aggression to the epithelium

bronchial airflow limitation and / or emphysema, for unbalanced relations protease / antiprotease. These effects are independent of the presence of pneumoconiosis and functionally similar to obstructive disease caused by tobacco and air pollution. The persistent inflammation around the airways can lead to the formation of localized areas of centrilobular emphysema by excessive release of proteolytic enzymes, explaining the changes and development of obstructive frames chronic airflow limitation (CAL) in exposed susceptible. It can not, however, rule out a possible synergistic effect of smoking and inhalation of inorganic dust, in the development process more aggressive tissue. In practice, the more functional defect observed in workers exposed to mineral dust is the airway obstruction.

3 METHODOLOGY

This protocol was drafted from a model suggested by the Technical Department of Occupational Health, Ministry of Health. The method used has been drafted as a reference point the experience of physicians invited to participate through discussion of form and content of the document and presentation of texts. These professionals have extensive experience in clinical care of workers with suspicion, or patients with pneumoconiosis at different levels of resolution, and also academic experience, through scientific publications, theses, texts and public holdings, being nationally recognized by his peers.

During the work we used the following sources:

- Texts published by the authors of the protocol.
- Texts from other authors published today in books, manuals and / or scientific articles.
- Books and publications of international organizations such as WHO and ILO.
- Brazilian legislation in force.

In formulating these recommendations were taken into account recent evidence regarding knowledge of the various pneumoconioses and research methods applicable. However, it should be noted, these recommendations are subject to change depending on the evolution of knowledge of their own illnesses and research methods. The flow of protocol development is outlined as follows:

4. 1 Definitions, occupational hazards and diagnostic methods

4.1.1 *Pneumoconiosis non-fibrogenic*

Definition: lung disease caused by exposure to fibrogenic dusts with low potential, also known as pneumoconiosis due to dust inert.

Examples: siderosis, baritose, estanose, pneumoconiosis coal plant, phosphate rock.

Occupations at risk:

electric arc welders, workers exposed to charcoal (production, storage and industrial use), workers of phosphate rock mining and bagging of barium and tin.

Diagnostic methods:

- History of occupational exposure to dust not fibrogenic.
- History of clinical symptoms with absent or with symptoms that are generally preceded by radiological findings.
- Chest plain radiography interpreted in accordance with 2000 ILO criteria.

Key features:

characterized by the accumulation of macrophages laden with particles arranged in macula, associated with reticulin fibers and collagen fibers and few expressed by small nodular opacities, associated or not with reticular, diffuse and bilateral. They usually occur after long-term occupational exposures. Respiratory symptoms are often lacking, and the dyspnea is the main one. Generally, diagnosis is an incidental finding or a periodic review.

Differential Diagnosis: miliary tuberculosis, sarcoidosis, paracoccidioidomycosis, histoplasmosis, other fungal infections, diffuse bronchiolitis.

4.1.2 *Pneumoconiosis fibrogenic*

4.1.2.1 *Silicose*

• *Chronic Silicosis*

Definition: pneumoconiosis caused by inhalation of free crystalline silica that manifests itself after long-term exposure, usually more than ten years, characterized by progressive fibrosis of the lung parenchyma.

Occupations at risk:

mining industry: surface and underground mining.

- Processing of minerals, stone cutting, crushing, grinding,

lapping.

- Manufacturing industry: ceramics, foundries that use sand in the process; glass.
- Abrasives, marble, granite cutting and polishing; cosmetics.
- Construction industry: tunnel boring, sanding walls, laying floors, cut stone.
- Mixed Activities: prosthetic; diggers of wells; artists; sand blasting operations.

Diagnostic methods:

- History of occupational exposure to dust containing free crystalline silica.
- History of clinical symptoms with absent or with symptoms that are generally preceded by radiological findings.
- Chest plain radiography interpreted according to the criteria of the ILO in 2000.

Key features: characterized by a reaction focal collagen deposition organized in concentric nodules of collagen fibers associated with the presence of bodies birefringent to polarized light.

Does not usually cause symptoms in the early stages and even moderate.

The dyspnea is the main symptom and physical examination, most often, does not show significant changes in unit respiratório. Expressa were radiologically by nodular opacities that begin in the upper parts.

Differential Diagnosis: miliary tuberculosis, sarcoidosis, paracoccidioidomycosis, histoplasmosis, other fungal infections, bronchiolitis diffuse.

•Accelerated silicosis or subacute

Definition: form of silicosis arising from occupational exposure to respirable dust with high concentrations of crystalline silica, manifesting between five and ten years following initial exposure.

Occupations at risk:

well diggers, stonecutters, and all other occupations at risk for exposure to silica in which there may be an intense exposure.

Diagnostic methods:

- occupational history of intense exposure to silica.
- Clinical history with respiratory symptoms earlier and limiting.

- Chest plain radiography interpreted according to the criteria of the ILO.

Key features: characterized by fibrotic nodules present, and often areas with focal lesions of acute silicosis. Present respiratory symptoms, particularly dyspnea

and cough. The radiological changes are rapid progression and is associated to an increased risk of comorbidities, especially tuberculosis and autoimmune diseases.

Differential Diagnosis: miliary tuberculosis, sarcoidosis, paracoccidioidomycosis, histoplasmosis, other fungal infections, diffuse bronchiolitis.

•Acute silicosis

Definition: A form of silicosis that occurs due to exposure to large quantities of dust from freshly fractured silica, characterized by diffuse alveolar damage and exudation of eosinophilic material in lipoprotein airspace and interstitial inflammation. Usually manifests itself after a few months or years of exposure.

Occupations at risk: operations with sand blasting, grinding stone.

Diagnostic methods:

- occupational history of intense exposure to silica dust for a short time.
- Clinical history with dyspnea rapidly progressiva. Radiografia simple chest interpreted according to the criteria of the ILO.

Key features: diffuse lung disease, quick installation, with respiratory symptoms and constitutional present, characterized by a anatomopatologicamente material deposition proteinaceous

intra-alveolar without interstitial fibrosis. It is a rare disease, occurring in situations of massive exposure to silica for periods ranging from a few weeks to four or five years, evolving rapidly to a lethal outcome (usually within one year of diagnosis).

Differential Diagnosis: Pulmonary alveolar proteinosis syndrome, adult respiratory distress, pulmonary edema.

4.1.2.2 coal workers' pneumoconiosis

Definition:

pneumoconiosis caused by inhalation of coal, its accumulation in the lungs and tissue reaction.

Occupations at risk:

miners mining front, detonators, transport and storage of coal in confined spaces.

Diagnostic methods:

- History of occupational exposure to dust generated in mining operations, transportation and storage of coal.
- Clinical history with respiratory variable, tending to asymptomatic in mild and moderate.
- Chest plain radiography interpreted in accordance with 2000 ILO criteria.

Key features:

characterized by formation of pigmented macules with peribronchiolar and perivascular reticulin deposits, sometimes associated with focal collagen organized in reaction to

form stellate nodules associated with the presence of bodies birefringent to polarized light. Does not usually cause symptoms in the early stages of the disease and intermediate. Occasionally, workers affected

develop progressive massive fibrosis.

Differential Diagnosis:

miliary tuberculosis, sarcoidosis, paracoccidioidomycosis, histoplasmosis, other fungal infections, diffuse bronchiolitis.

4.1.2.3 Pneumoconiosis in mixed dust

Definition:

pneumoconiosis caused by exposure to mineral dust with low content of crystalline silica, as in exposure the dust of mica, kaolin, sericite, marble, in processes with use of abrasives in foundries and in some cases the ceramic industry.

Occupations at risk:

workers in mining and processing silicates, such as mining, milling and use of mica, kaolin, sericite, feldspar, potters, deburring.

Diagnostic methods:

- Occupational history of intense exposure to dust with a high content of silicates.
- Clinical history with respiratory variable, tending to asymptomatic in mild and moderate.
- Chest plain radiography interpreted according to the criteria of the ILO in 2000.

Key features:

reaction is characterized by focal collagen organized in stellate nodules and diffuse interstitial fibrosis associated with the presence of

bodies birefringent to polarized light. Usually occur after long-term occupational exposures.

Differential Diagnosis:

miliary tuberculosis, sarcoidosis, paracoccidioidomycosis, histoplasmosis, other fungal infections, diffuse bronchiolitis.

4.1.2.4 Asbestos-related Diseases

• *Asbestosis*

Definition:

pneumoconiosis consequent to inhalation exposure to dust containing asbestos fibers.

Occupations at risk:

workers in mining and processing of asbestos (manufacture of asbestos-cement products, friction materials, fireproof fabrics with asbestos gaskets and seals, special paper and cardboard) and consumption of products containing asbestos.

Diagnostic methods:

- History of occupational exposure to dust with asbestos fibers.
- Clinical history with respiratory variable.
- Chest plain radiography interpreted according to the criteria of the ILO in 2000.
- High-resolution computed tomography.

Key features:

characterized by diffuse interstitial fibrosis in lung inflated, away from areas of tumor or other lesion associated with the presence of two or more asbestos bodies per cross sectional area of 1cm²

When not found to be the counting of asbestos fibers, which should be in the range expected for asbestosis according to the reference laboratory for analysis under way. Exertional dyspnea and dry cough that may progress to dyspnea at rest, hypoxemia and cor pulmonale. The radiological changes are characterized by the presence of irregular opacities predominating in the lower fields,

and often associated with pleural plaques.

Differential Diagnosis:

emphysema, usual interstitial pneumonia, collagen diseases, lymphangitis carcinomatosa.

- Pleural disease by asbestos

Definition:

fibrosis of the parietal pleura and / or visceral as a consequence of exposure to dust with asbestos fibers.

The asbestos-related pleural abnormalities can present as circumscribed pleural thickening (pleural plaques) or diffuse, with or without calcifications, pleural effusions, rounded atelectasis and streaks pleuroparenchymal fibrosis.

Occupations at risk:

workers in mining and processing of asbestos (manufacture of asbestos-cement products, friction materials, fireproof fabrics with asbestos, gaskets and seals, special paper and cardboard) and consumption of products containing asbestos.

Diagnostic methods:

- History of occupational exposure to dust with asbestos fibers.
- Clinical history with respiratory poor.

Pleural plaques are usually asymptomatic. The diffuse pleural thickening, when moderate or extensive courses with symptoms of functional constraint - dyspnea. Pleural effusion may be asymptomatic or present with symptoms of chest pain, fever, dyspnea on exertion. • Chest plain radiography interpreted according to the criteria of the ILO in 2000.

- High-resolution computed tomography.

Key features:

The circumscribed pleural thickening or pleural plaques are focal areas of irregular fibrosis, almost devoid of vessels and cells, as well as signs of inflammatory reactions that occur primarily in the parietal pleura, and are more often seen in regions posterior lateral chest wall and also in the diaphragm and mediastinal regions.

It is the most common disease caused by inhaling asbestos fibers. The diffuse pleural thickening is a disease that affects the pleural, does not present a specific format, varies in width between 1mm and 1cm or more, is usually bilateral and frequently is associated with fibrotic bands that enter the parenchyma. It is less specific exposure to asbestos and may appear as a sequela of an inflammatory reaction

caused by other diseases. Pleural thickening may extend to areas of interlobar and interlobular septa, usually consequent pleural effusion, causing a twisting of the area of lung parenchyma, which is coiled and showing atelectasis, leading to a round image, known as rounded atelectasis. Pleural effusion by asbestos can occur at any time of the exhibition and presents

characteristics of exudate. It is often bloody, with presence of leukocytes, mesothelial cells and eosinophils. It is usually asymptomatic but may present with pleuritic pain and fever. It can last several months, be unilateral or bilateral and appeal.

Differential Diagnosis:

pleural plaques alone, particularly diaphragmatic plaques are highly suggestive of exposure to asbestos. Wall panels are the main differential diagnoses subpleural fat, shadows muscle and rib fracture. The diffuse pleural thickening can have other causes, such as a sequela of tuberculosis, surgery, chest trauma or drug reaction. The differential diagnosis of pleural effusion is important to remember tuberculosis pleural effusion and neoplastic diseases.

4.1.2.5 Pneumoconiosis in abrasive

Definition:

in this protocol is defined as the pneumoconiosis caused by inhalation exposure to dust abrasives: aluminum oxide or corundum (Al_2O_3) and silicon carbide or carborundum (SiC).

Occupations at risk:

workers in the production of abrasives, in finishing operations in foundries and metal plants in general, sharpening tools milling and grinding of scrap.

Diagnostic methods:

- Occupational history of intense exposure to dust containing alumina or carborundum, as well as dust derived from the material being worked.

- Clinical history with respiratory variable, tending to asymptomatic in mild and moderate.

- Chest plain radiography interpreted according to the criteria of the ILO in 2000.

Key features:

shows similar characteristics to those of mixed dust pneumoconiosis.

Differential Diagnosis:

miliary tuberculosis, sarcoidosis, paracoccidioidomycosis, histoplasmosis, other fungal infections, diffuse bronchiolitis.

Pneumoconiosis 4.1.2.6 for hard metals

Definition:

pneumoconiosis caused by exposure to dust from metal alloys composed of tungsten and other heavy metals such as titanium, tantalum, niobium and vanadium on the property associated with the cobalt binder.

Occupations at risk:

workers in the production of tools and pieces of hard metals in sharpening tools made of alloys and other Widia, special wheels and

dentures.

Diagnostic methods:

- History of occupational exposure to dust containing alloys hard metals.
- Clinical history started with progressive dyspnea after a certain exposure time (variable from months to years).
- Chest plain radiography interpreted according to the criteria of the ILO in 2000.
- High-resolution computed tomography of thorax.
- bronchoalveolar lavage differential cell counts for research.
- Discussion of need for biopsy.

Key features:

characterized by a desquamative interstitial pneumonia with giant cells. Coexists with symptoms of fatigue, dyspnea, early, cough dryness, pain, chest tightness and other constitutional symptoms. With progression of the disease may appear fever and weight loss. In general, the symptoms appear after a period of "sensitization" variable from months to years.

Differential Diagnosis:

emphysema, usual interstitial pneumonia, asbestosis.

4.1.2.7 Pulmonary by beryllium

Definition:

lung disease caused by inhalation of fumes or dusts of beryllium salts.

Occupations at risk:

workers in the aerospace industry, nuclear power industry, manufacturing and use of special grinding wheels and special alloys in dental prosthetics.

Diagnostic methods:

- History of occupational exposure to dust containing beryllium.
- Clinical history started with progressive dyspnea after a certain exposure time (variable from months to years).
- Chest plain radiography interpreted in accordance with 2000 ILO criteria.
- CT scan of chest high-resolution.
- bronchoalveolar lavage differential cell counts for research.
- Discussion of need for biopsy.

Key features:

characterized by manifest itself in two forms: a table of acute irritation of the tracheobronchial tree and may lead to chemical

pneumonitis, with consequent hypoxia and secondary fibrosis and chronic disorder characterized by granulomatous pulmonary and systemic involvement secondary to chronic exposure to low doses, Call for Pulmonary Disease Beryllium or BDP. The lag time is on average 10 to 15 years and may occur years after exposure has ceased. The DPB is associated alveolitis characterized by accumulation of lymphocytes and macrophages within alveoli and interstice adjacent to granuloma formation not DRINKS, sarcoid like, suggesting the pathophysiological mechanism involving immune response by delayed-type hypersensitivity. The main symptoms are chest pain, cough, fatigue, weight loss and arthralgia, which may present with adenopathy, skin lesions, hepatosplenomegaly and clubbing.

Differential Diagnosis:

Sarcoidosis, tuberculosis, paracoccidioidomycosis, histoplasmosis.

Hypersensitivity pneumonitis

Definition:

The hypersensitivity pneumonitis (HP) is not a pneumoconiosis itself. Also known as extrinsic allergic alveolitis, is a group of lung diseases resulting from sensitization by inhalation exposures to applicants of antigenic particles derived from organic matter and some chemicals (eg, phthalic anhydride and toluene diisocyanate, TDI), both in workplace and in others.

Occupations at risk:

workers in animal husbandry, transport processes, loading, unloading and storing agricultural, handling and other chemicals.

Diagnostic methods:

- History of occupational exposure to organic dusts with allergenic potential.
- Clinical history started with progressive dyspnea after a certain exposure time (variable from months to years).
- Chest plain radiography interpreted according to the criteria of the ILO in 2000.
- High-resolution computed tomography of thorax.

The temporal relationship between exposure and suspected clínicosão framework of fundamental importance to establish a "causal link" as in pneumoconiosis, which are diseases of large períodode induction (latency). Activities outside the workplace, comohobbies should also be listed. There is a "aprendizadoformal" in occupational history - we need to acknowledge that simplesindagação of "profession" is insufficient and uninformative regarding the respiratory risk exposures. So the curiosity of the professional who investigates a suspected case study and practical experiences are basic ingredients to get good data. Occasionally it is necessary that the place of

work to be played for a correct understanding of the exhibition.

4.2.2 Questionnaire on respiratory symptoms

The questionnaires on respiratory symptoms are commonly used in evaluation of groups, but can be used in individual assessments as an additional tool in history.

The main advantage of the questionnaire is to standardize information and the possibility of gradation of symptoms. To be a useful

tool it must obey the principles of validity and reliability (repeatability). In practice, two questionnaires are used: the questionnaire

chronic bronchitis and the Medical Research Council questionnaire on respiratory symptoms of the American Thoracic Society. The latter can be completed by the interviewee. Both investigate the cough, phlegm, dyspnea, wheezing and smoking.

4.2.3 Imaging methods

The reference method for the analysis of conventional radiographs of the chest is the Radiological Classification of the ILO, whose latest release is 2000. It allows X-rays are interpreted and encoded in a standardized way, by using standard comparative radiographs and appropriate log sheets. The radiological changes

are summarized with information about the patient's identification and radiography, quality of the plate, changes in lung parenchyma, pleural changes and symbols, which denote changes associated or not with pneumoconiosis.

A roadmap on Radiological Classification of the ILO is at Annex B. A hint of Radiological Leaf Reading is at Annex C. The frequency of radiographs is dictated by labor laws. It is necessary for the professional who plays the examinations has

specific training and appropriate to do so, since a diagnosis of occupational lung disease is accompanied by procedures and legal consequences that affect the life of patients. The radiological changes of pneumoconiosis are more frequent:

Exposure to silica dust and that presents with nodular opacities:

images are the expression of the accumulation of dust-laden macrophages (macules) in pneumoconiosis caused by dust or no fibrogenic fibrotic nodules, as in silicosis.

Are micronodular opacities, types p, q, r, usually starting in the upper third

of both lungs. In advanced cases, there may be images of calcified mediastinal lymph nodes, known in English as eggshell (symbol **s**), **distortion of intrathoracic structures (symbol di)** conglomeration of nodules (symbol **ax**). **In fibrogenic pneumoconioses,**

radiological changes may progress independent of continued exposure. The progression of lesions can lead to large opacities, classified as A, B or C, according to

ILO criteria.

Exposures to asbestos:

plaques and diffuse pleural thickening are better visualized in the lower halves of the side walls of the chest radiographs in posteroanterior earlier. The radiological appearance depends on the location and density of the plates can be viewed tangentially as one or more opacities forming a sharp interface of the wall with pulmonary parenchymal opacities or how that overlaps with the parenchyma, without the appearance of an anatomical structure intraparenchymal.

The oblique projections can help the visualization. The plates need to be differentiated from extrapleural fat (especially in obese patients). In case of unilateral thickening, you should make the differential diagnosis of reactions with rib fractures and muscle shadows.

When viewed from the front (face on), chest X-ray, they can simulate pulmonary nodules (if single) or pulmonary fibrosis (for multiple and small), which may hinder a proper reading of amendments to the lung parenchyma. Calcifications in a thickening allow facilitation in identification.

Radiology of asbestosis is characterized by the presence of small irregular opacities, types s, t, u, usually bilateral and commonly involving the lower lobes. Are also occasionally noted parenchymal bands (symbol **bp**) and septal lines (symbol **kl**). **With the progression of fibrosis, there may be signs of reduced volume and honeycombing. Other symbols are common radiological thickening of the horizontal scission (pi) the lack of cardiac contours (ih) and diaphragmatic (id). Once these changes are described in specific, the presence of pleural plaques are associated with strong evidence of exposure to asbestos. Currently, high-resolution computed tomography (HRCT) has been used with greater consistency in the investigation of suspected cases.**

HRCT is superior to conventional radiography in detecting lesions pleuropulmonary caused by exposure to asbestos, but until now, that course with pneumoconiosis in nodular opacities such as silicosis, for example, there is no consistent evidence in the literature, sufficient to be considered as a method of choice in diagnosing early stages of the disease.

The technique recommended for HRCT is described in Annex D. The tests should be done in the prone position, to eliminate the gravitational effect at the bases of the initiation site of fibrotic changes caused by asbestos. The cost of TCARs is still prohibitive for the examination of his appointment as medical control periódico.As

tomographic present in pneumoconiosis are summarized below:

In patients exposed to silica, coal dust and other evolving with nodular opacities: presence of nodules and also along the axial interstitium including fissures. In cases of silicosis, the disease onset usually affects the posterior portions of the upper lobes. With the evolution of the process, you may notice conglomeration of injury, usually in the upper lobes.

In patients exposed to asbestos: pleural plaques are seen on HRCT in the parietal, diaphragmatic and mediastinal structure as a radiological density similar to muscle, defined as thickening, with or without calcifications. When the diaphragm reaches the pleural thickening due to the cutting plane of the CT scan, your view on HRCT can be impaired when it is not calcified. HRCT allows to differentiate between the thickening of intrapulmonary lesions and extrapleural fat. The CT changes associated with asbestosis are:

1. Intralobular interstitial thickening.
2. Interlobular septal thickening.
3. Subpleural lines 1 to 10 cm, parallel to the pleura, which are usually with septal lines.
4. Parenchymal bands from 2 to 5cm, touching the pleural surface.
5. Frosted glass, changes that persist when changing the patient position, corresponding histologically to thickening of the alveolar walls and interlobular septal involvement. Possibly reflect alveolitis.
6. Lobular disarray subpleural.
7. Bronchiolectasis or traction bronchiectasis.
8. Honeycombing, cystic spaces small, smaller than 1 cm in diameter, with slight wall thickening.

4.2.4 Lung Biopsy

Occasionally, exhaustion of noninvasive diagnostic methods, lung biopsy may be indicated in the following situations:

1. Radiological abnormalities consistent with exposure, but:
 - occupational history with uncharacteristic or absent;
 - with a history of exposure to dust or other unknown agents;
 - insufficient exposure to cause changes observed;
 - radiological appearance inconsistent with the type of exposure reported.
2. In cases of litigation, after disagreement between at least two

players due to familiarizados/credenciados⁵ radiological interpretation of the International Classification of Radiographs of Pneumoconiosis ILO. In these cases, we recommend the implementation of abnormality, also interpreted by an experienced professional in the method, before the definition of lung biopsy. A lung biopsy should be performed in-service trained and performed by a pathologist with expertise.

4.2.5 Functional tests

The pulmonary function tests are essential in the investigation of occupational respiratory diseases that affect the airways, as well as the establishment of disability in patients with pneumoconiosis. In contrast to occupational asthma, pulmonary function tests are not applicable in the diagnosis of pneumoconiosis. Spirometry is the most commonplace of functional evaluation. It takes a fast, easy and low cost. In occupational terms, the main indications are 1. Evaluation of workers with respiratory symptoms.

2. Evaluation of respiratory dysfunction and disability.

3. Longitudinal follow up of workers exposed to respiratory hazards.

Its usefulness in individual assessments (clinical) of workers seeking medical attention for respiratory problems is similar to routine clinical practice. Standardization of spirometry, the items relating to equipment, technique and technical quality control and interpretation, shall meet the criteria of the Brazilian Consensus on Spirometry. In occupational terms the "healthy worker effect" is clearly present in certain economic sectors. This is a phenomenon of selection that concentrates workers with physical skills necessary to perform certain functions, for example, underground mining. In general, workers with altered pulmonary function or respiratory symptoms tend not to remain in office

high physical demand. Therefore, it is common to find normal spirometry groups exposed to respiratory hazards, and even in patients with pneumoconiosis. Some situations require greater sophistication in the exploration of pulmonary function, especially the assessment of respiratory dysfunction and disability compensation for purposes of social security and civil remedies. The study of diffusion of carbon monoxide (CO) and assessment of exercise capacity distinguished by its ability to better assess the complaints of dyspnea, not always expressed by spirometric changes. These methods are usually available in reference services in pulmonology.

4.3 Diagnosis of pneumoconiosis

The diagnosis of pneumoconiosis and pleural abnormalities by asbestos based on the triad: a. Consistent work history.

b. Latency compatible.

c. Changes compatible image. Changes are considered compatible image:

- Chest radiograph with radiological findings $\geq 1 / 0$ (radiological characteristics are described in subsection 4.2.3 and Annex B) and / or the presence of pleural abnormalities.

- High-resolution computed tomography with changes consistent with exposure above. In cases of exposure to silica dust and mixed dust from coal, as well as some non-fibrogenic dusts the typical consist of centrilobular nodules of varying density and profusion, and nodules along the axial interstitium of the lung. CT changes are considered definitive of fibrosis by asbestos exposure: the presence of bronchiolectasis or traction bronchiectasis and honeycombing. If these changes are not present, there must be at least three types of changes (1-6), described in subsection 4.2.3, in more than one level of court and bilateral. Cases of unilateral changes consistent with fibrosis are rare, and deserves further evaluation. The diagnosis of a case of pneumoconiosis and / or by asbestos pleural disease therefore requires the integration of occupational history, exposure time and latency and compatible radiological findings according to the criteria of the International Classification of Radiographs of Pneumoconiose/2000 ILO. The interpretation of chest radiography should be performed by a medical professional before undergoing a qualification / training. For confirmation of the radiological abnormalities compatible, it is essential that, beyond training, the reader

has at its disposal a set of standard radiographs of the ILO, in order to make comparative radiological readings and classify the radiographs according to criteria established in relation to high-resolution computed tomography of the chest, considering the cost of the examination, shall be made at a place that is guaranteed good quality radiographs and training of professional

responsible for interpreting the images obtidas⁷. As indications of biopsy are summarized in subsection 4.2.4. It must be ensured that the interpretation of results is made by pathology services and reference by experienced pathologists in the reading of slides with suspected pneumoconiosis.

A ranking of the diagnosis takes into account the resolution capacity of the health system. It is possible that in certain places, the final diagnosis can be done in Primary Care Units. When there are no possibilities of obtaining good quality radiological and trained readers,

the patient should be referred to a reference unit secondary or tertiary.

4.4 Treatment

For all pneumoconioses mandatory expulsion is no indication that exposure to causou.Tratamento medication is indicated only in pneumoconiosis with pathogenesis-related hypersensitivity response, such as the lung by cobalt, the lung and the beryllium hypersensitivity pneumonitis. In these cases, in addition to binding and final removal from exposure, the glucocorticoid is indicada.Nos not fibrogenic pneumoconiosis cases, removal may eventually produce a reduction in the intensity of radiographic opacities.

the settled, and spray mist of water at the point of production of dust. Two classical measures in this type of control are located exhaustion, which should be installed against the flow of inhaled worker in his job, and general ventilation of the environment as a whole. Other basic measures of industrial hygiene are the total or partial enclosure of dust-producing process, with external operation, trying to isolate polluting processes, and changes in layout. Replacing matérias-primas/produtos preventive measures are of great importance, as the use of other abrasive sand blasting operations and the use of alternative fibers in asbestos-cement products, friction materials and others. Substitutes should have a known toxicity profile in order not to incur the replacement of certain other similar risks. The industrial fumes to the outside should be subjected to processes that minimize or eliminate its impact on the environment and the neighborhood. The individual respiratory protection should be used in operations where respiratory protection measures for collective are insufficient to control inhalation exposure. The use of respirators must fit the type of aerosol generated and be part of a Respiratory Protection Program. Respirators must be of good quality, efficiency, show good adaptation to the face of the worker, to have periodic maintenance, cleaning and replacement filters when needed. Wash contaminated clothing containing dust must be made by the company to avoid the risk of contamination of their educational familiares.Ações are of fundamental importance in primary and secondary prevention of pneumoconiosis. It is not uncommon ignorance of risk in work environments with risk of exposure to inhaled dust. Information on risks involved in different processes must be priorities, both for employers and employees. Prevention programs within companies have a greater chance of success with the active participation of the involved segments. Based on principles of screening, the medical control seeks to identify the disease in its latent state, when some kind of intervention can

halt, reverse or slow the onset and progression of abnormal physiological conditions. The application of standard routines, such as respiratory symptoms questionnaire, physical examination, radiographs and spirometry journals are intended to identify such cases. The medical control, in that sense, despite being called "secondary" serves as a privileged source of information feeding the control "primary" industrial hygiene, indicating the need for changes in the productive process, collective protection, such as enclosure, exhaust ventilation and use of personal protective equipment, in the most specific exception. The types of exams and its frequency should be based on literature and clinical common sense. The current issue of NR-7 CLT brought important changes that allow more freedom in developing prevention programs, encouraging epidemiologic methods.

The largest annual basis or to perform chest X-rays in environments at risk for inhaled dust, contained in several national and international standards, can not be scientifically grounded, as is known, for example, the average latency for the appearance of certain pneumoconioses such as silicosis and asbestosis. Even

in the case of fibrogenic dust, the proposal to hold annual chest X-rays only make sense for early detection of cases after years of exposure, except in cases demonstrating atypical clinical course, especially in suspected acute or subacute silicosis. However, for labor, firms that fall in the risk of exposure to mineral dust should comply with instructions contained in NR7. Also in relation to secondary prevention, we must remember the increased risk of tuberculosis in individuals exposed to silica, even if they are removed from exposure, as well as the increased risk of lung cancer in workers exposed to asbestos and silica.

Data that lead to suspicion silicotuberculosis is a rapid progression of lesions, cavitations, conglomerates and large opacities besides the constitutional symptoms such as asthenia, weight loss and persistent low fever. In 1996, the International Agency for Research on Cancer (IARC) has classified silica as Group I, ie, a substance described as carcinogenic to humans. There is an excess risk in silica-exposed, predominantly in chronic silicosis. Individuals exposed to asbestos pleural or parenchymal involvement without the higher risk of developing cancer compared with unexposed, constituting a risk group and should be subject to monitoring.

The presence of pleural thickening and / or asbestos and is associated with an increased risk for cancer, probably reflecting a greater burden of exposure, although there is no safe tolerance limits for carcinogens. The presence of pleural thickening and / or asbestosis is

associated with a higher risk, probably reflecting a greater burden of exposure, although there is evidence on the existence of safe tolerance limits for carcinogens.

4.6 Conduct in patients with pneumoconiosis

Diagnosed cases should be treated as "sentinel case" and should be duly notified and trigger actions integrated surveillance, with the objective of detecting other cases not yet diagnosed within the environment generating the disease, and adoption of preventive measures and protection to exposed workers, as described in item 4.4.

As a general rule, workers with pneumoconiosis should be removed from exposure that caused the illness, because continued exposure leads to a worsening of the condition. However, it is recommended that professionals responsible for directing has common sense in judging whether the activity and the conditions which caused the disease persists at the time of establishing the practice. It is possible that exposure does not, by process change or total effectiveness of preventive measures. Pneumoconiosis is notifiable diseases in the National Health System, regardless of their working ties. Workers in the formal market also mean, by notification in the Work Accident Communication (CAT), which is a document of the Ministry of Welfare and Social Security. The CAT can be issued by the company by the union or any nprofessional health involved in the process of investigation. With this document, the affected worker shall undergo a medical assessment by Social Security to evaluate the "causal" and "disability", the criteria used in judging the right to social security benefit. A "failure" is differentiated from "dysfunction," according to the settings below:

Dysfunction: is the reduction of respiratory function and are usually assessed by pulmonary function tests during rest and exercise and questionnaires assessing symptoms, particularly dyspnea. Task is primarily medical.

Disability: is the overall effect of the dysfunction in the patient's life expressed by the inability to adequately perform a task at work or in daily life due to malfunction. Failure is not only related to medical conditions, but involves more complex factors such as age, sex, anthropometry, education, psychological status, socioeconomic status and type of energy requirement of the occupation, becoming a medical administrative assignment. The diagnosis of pneumoconiosis needs to be accompanied by an inability to work or need for change of function

so that the worker is entitled to social security benefit. However, even without dysfunction, the occurrence of disease is sufficient to queo

employee filed a lawsuit for injury at common law. Workers suffering from pneumoconiosis, and removal procedures of exposure, notification and pension administration, when the law (formal workers) should be monitored regularly by clinical and imaging, and, where possible, functional assessment by spirometry biennials. It should also be guaranteed the attendance and conduct of examinations

Additional whenever the situation of the patient so desired (appearance of symptoms, episodes of decompensation, association with other diseases). Occupational Lung Diseases

DERMATOSES

The occupational dermatitis represent considerable portion of disease professionals. Its prevalence is difficult and complex evaluation. Many of these dermatoses is not enough statistics and even the knowledge of experts. Many are self-treatment, others are treated in the ambulatory of the company. Some come to the clinician and the specialist doctors in the consortia that provide assistance under an agreement with the National Social Security Institute (INSS). Only a small share of these dermatoses comes to specialized services. Skin diseases caused by physical, chemical and biological agents from occupational exposure and working conditions are responsible for the discomfort, pain, itching, burning, and other psychosomatic reactions which generate even loss of job. These conditions are inherent in the work organization that seeks to achieve the goals of high productivity and product quality, with the sizing of workers and material resources provided by companies without the criterion of quality of work life is indeed taken into account. The organization of work, without considering the human factor and its limits, is structured in different hierarchical levels, with the characteristic inflexibility and high-intensity work rate, pressure for productivity and inability to control by workers. **One of the steps required for this process is knowledge of** real situation of workers, regardless of their insertion

labor market. Taking responsibility for comprehensive health care worker, the NHS must to be a rich and comprehensive **source of information and as executor of interventions to prevent** at all levels of health care. The data obtained through records of attendance, once formatted and computerized, will be instrumental in the short term, give a more real to the health of workers in our country That effort now developed that can generate knowledge levels aimed at mobilizing resources to interact with the factors causing diseases in workers; to

minimize them as much as possible and neutralize them completely.

SCOPE

2.1 Disease or condition

It's all change in the mucous membranes, skin and its annexes, which directly or indirectly caused, conditioned, maintained or exacerbated by agents present in an occupation or in the workplace (ILO, 2001).

2.1.1 Causes of occupational dermatitis

Two main groups of factors can be listed as conditioners of occupational dermatitis:

- indirect causes or predisposing factors;
- direct causes, are composed of biological, physical, chemical, existing environmental and laughed acts directly on the seed coat, either causing or exacerbating preexisting dermatosis (BIRMINGHAM, 1998).

2.1.2 Indirect causes or predisposing factors

•Age:

•Young workers are less experienced, more affected by costumanser act with less caution in manipulação chemicals potentially perigosospara skin. On the other hand, the integument is not yet adapted to contatante to produce the espessamentoda stratum corneum (Hardening) tolerance or adaptation to the agent. (LAMMINTAUSTA; Maibach, 1990).

•Gender:

•men and women are equally affects the. However, women have greater impairment in hands and may be followed less severe and more rapid remission (PATIL; Maibach, 1994; Meding, 2000). Women, in general, have better outcomes in their acne (Nethercott; Holness, 1993).

•Ethnicity:

•people of the yellow race and the black race are more protected against the action of sunlight that people were white, black with responses in keloids more often than whites. There are racial differences in the penetration of chemical agents and other substânciasna skin. Several studies have shown that the black race has agents penetration lower than Caucasians and that the stratum corneum of the black race has a greater number of layers and spontaneous peeling of this layer is two and a half times greater than in white and yellow (Berardesca; Maibach, 1988).

•Climate:

•temperature and humidity (HOSO et al, 2000) influenciando appearance of skin lesions such as pyoderma, fungal infections, miliaria. The outdoor work is often subject to the action of sunlight, insect bites, contact with plants, exposure to rain and wind, bemcomo agents for various potentially dangerous skin.

•Morbid history and concomitant dermatoses:

•patients with atopic dermatitis or atopic diathesis are more susceptible to the action of irritants, especially alkaline, and can develop contact dermatitis caused by irritants and do not tolerate well the humidity and high temperature environments; activity in patients with dermatitis (nummular eczema, eczema irritative, dermatitis Plant, psoriasis, lichen planus, etc.). are more likely to develop acne or occupational acne had worsened in their working environment, specific protective are neglected.

•Working conditions:

•The work in standing position, in susceptible workers may lead to the onset of stasis dermatitis, varicose veins or aggravate existing ones.

•Presence of vapors, gases and dust above the limits of tolerance may be a predisposing factor, as well as the lack of lighting, proper ventilation and adequate toilets and showers clean and close to workplaces.

•Failure to use adequate protection or incorrect use or the use of Personal Protective Equipment (PPE) of poor quality and a failure by the standards of worker health and safety standard for the activity they perform, may play important role in the onset of occupational dermatitis.

2.1.3 Direct causes

Biological, physical and chemical. May cause occupational dermatitis or act as triggering factors, or aggravating competitors. The most common biological agents: bacteria, fungi, yeasts, viruses and insects.

•Physical agents. The main ones are: non-ionizing radiation, heat, cold, electricity.

•Chemical agents. The main ones are:

1. Irritants →cement, solvents, cutting oils, detergents, acids and alkalis.

2. Alérgenosaditivos rubber, nickel, chromium and cobalt as contaminants in the cement, resins, used in topical treatment of dermatoses.

2.2 Admission, diagnosis, treatment and prevention

Admission will be structured from the proper forms that include objective data of the worker. The employee, after completing a form, should be examined in the appropriate location with adequate light. This procedure is described in Annex B. Diagnosis or diagnosis following procedures which, if done consistently will lead to hit rates for the possible causative agents and occupational link.

2.2.1 Diagnosis: how to identify cases of dermatoses occupational

For the diagnosis and establishment of appropriate conduct of occupational skin diseases, suspected or confirmed, it is important to consider

the following: Clinical picture, history of occupational exposure, observing

correlation between the onset of symptoms and the onset of exposure, as well as the location of lesions in areas in contact with the suspected agents. Improvement with the removal and worsens with the return to work. this positive skin patch, where a contact dermatitis caused by sensitization.

Table 1 - Diagnosis of contact dermatitis: allergic and irritant.

The clinical picture is compatible with contact dermatitis? Occurs in the workplace exposure to irritants or potentially allergenic? Nexus exists between the onset of acne and period of exposure?

(Concordance anamnestic)

The lesions are located in areas in contact with the suspected agents?

(Concordance topographic)

There are better with spacing and / or worsened by the return to the same

activity? You can delete the non-occupational exposure as a causal factor.

It is possible through the patch tests to identify the likely agent causal?

Note: five of these positive alternatives, are strongly suspected occupational dermatosis. The diagnosis of occupational dermatitis (Fisher, 2001; BIRMINGHAM,

1998) is done with relative ease, except in some borderline cases where it becomes difficult to do so. Some aspects are very important to

obtain an accurate diagnosis. Among them we quote:

1. patient identification;
2. interview;
3. physical examination;
4. diagnosis;
5. differential diagnosis;
6. exams;
7. visit to the workplace;
- b) occupational Anamnesis

The occupational history, as occurs in all specialties

Medical, is an important tool for diagnosis. A good occupational history will lead us to the possible etiologic agent. For this it is necessary to have proper form and a prepared script, where the data required to be reported.

Personal history. Atopy, or family, previous skin disease.

Personal or family history of asthma, rhinitis, atopic dermatitis. These pathologies, have specific meanings making the skin of these patients more susceptible to attack by various agents.

c) Physical Exam

Physical examination is of paramount importance to assess the type, location and extent of injuries presented seated. Note: If necessary, refer to Appendix B - Examination dermatológico. Toda the skin should be examined by noting, if possible, a puppet drawn on paper itself, the location of the lesions. In dermatology, occupational injuries occur more frequently in the hands, forearms, arms, neck, face and legs. However, in some cases, all skin can be achieved.

If possible, can be idealized form suitable for being recorded their findings on physical examination. This contains data that can guide treatment and prevention of further recurrences.

To record:

Detailed Description of injuries: injuries noted on the doll.

Location Symmetry Color Form

There is impotence of the affected area? ☐ Yes ☐ No

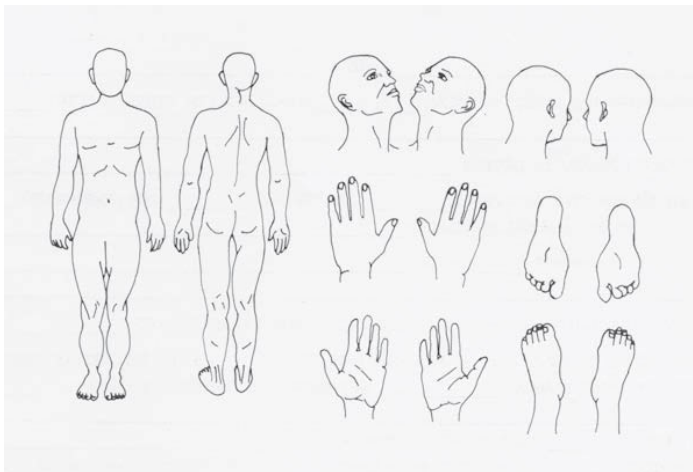


Figure 1 - Report on the design areas with dermatoses

d) Diagnostic hypothesis

When formulating the HD becomes important to check whether there is a causal relationship.

This dermatitis are occupational dermatitis more frequentes. Estima that, together, the allergic contact dermatitis and irritant contact dermatitis represent about 90% of cases of occupational dermatitis. Although in most cases do not produce pictures as serious, are often responsible

by discomfort, itching, wounds, trauma, aesthetic and functional changes that affect the social life and work.

2.2.2 Differential Diagnosis

Considerations:

Many workers with skin diseases are referred to serviços especializados with the primary diagnosis of occupational dermatosis, although many of them correspond to non-occupational skin processes. In this situation, if necessary, one should resort to the expert who must establish the correct diagnosis of acne.

Non-contact dermatitis, occupational allergic or irritant may mimic allergic dermatitis and irritant occupational contact. It is a true contact dermatitis. However, a good history can show us that it is not of occupational etiology and therefore has no causal link. One can also infer that an occupational contact dermatitis may be aggravated by activities of the weekend, such as reform of the residence, repairs, and other paintings. Other skin conditions can often raise doubts about the causal relationship,

such as psoriasis, herpes simplex and herpes zoster, idiopathic vesicular reactions by the presence of ringworm on the feet mícides (Dyshidrosis eczema), nummular eczema and skin reactions to drugs, etc.. If in doubt, refer the affected worker to a specialist.

2.3 Investigations: histopathology, patch tests and other

2.3.1 Laboratory tests

Are rarely requested.

2.3.2 Histopathology

Preferably, it should be requested by the specialist. Patch tests are required in cases of suspected allergic contact dermatitis.

2.3.3 Test of contact - concept

It is done in a biotest with the back of the patient containing a battery of allergens known as the standard battery and more suspected allergens handled, and those used to treat acne. The contact test is a method for studying allergic to well established rules and fundamentals. Through the test Contact Dermatitis, irritative can differentiate Contact (DIC) of Allergic Contact Dermatitis (ACD). Based on test results, we can guide the patient to avoid further contact (s) agent (s) complained (s) with skin and institute appropriate preventive measures when new contacts are imperative (FISHER, IRMA KIHLMAN, 1989; AMERICAN CONTACT DERMATITIS SOCIETY, 1994).

The contact test with allergens should be prepared in good quality and provenance. The mixture of the allergen with the vehicle should be as homogeneous as possible and obey the appropriate technology. The tests should have high quality and has good sensitivity and specificity (FISHER; Maibach, 1990). The sensitivity of the test is directly related to its ability to identify whether the patient introduced by contact allergy. Specificity is the ability of the test has to discriminate a true allergic reaction to another non-allergic. Precision or accuracy is the sum of sensitivity and specificity.

Low sensitivity of the test can express a false negative.

Low test specificity can express a false positive.

Relevance: we say that the allergen from a positive test has significance when it is related to acne. We took the accused in allergen testing and courses to cure acne or insignificant improvement of the clinical (Podmore, BURROWS, BINGHAM, 1984).

Table 1 - Interpretation of the contact test:

- Mono awareness: a positive allergen is responsible for Allergic Contact Dermatitis (ACD). Your total withdrawal leads to healing of acne.
- Poly awareness: multiple positive allergens, the withdrawal of all leads to a cure or significant improvement.
- Poly awareness: Several positive allergens, the withdrawal of all does not cure or improvement of clinical status. In this case check:
 - a) There are no other allergens tested and were not detected by occupational history?
 - b) factitious dermatitis, dermatitis artefacta, self-injury. The worker knows ways of keeping your acne active, to prevent their dismissal or loss of benefits.
 - c) worker can eventually run away from temporary jobs (beak) to improve the income and activity in that contact sensitizers or irritants that impede good deed for the proposed treatment.

Contact test with standard battery and suspects

The contact test should always be done with a standard that will apply to all suspects with Allergic Contact Dermatitis (ACD). Using standard tables of concentration by a study group of contact dermatitis of the Brazilian Society of Dermatology. Besides the standard, test substances referred the patient by means of anamnesis, which must be well prepared and thorough. **The suspected allergens, obtained by previous history should be tested with standard battery.**

All precautions must be taken when testing substances of unknown composition. The test should be offered whenever there is suspicion or evidence that it is an Allergic Contact Dermatitis (ACD). The test should be performed by previously trained staff and its reading and interpretation done by professional

Note: The physician involved in health care worker should know the work environment of each activity. For this we suggest that it has at least one or two days per month to meet the workplace in order to better understand the hardships that a worker may suffer in that specific activity. During these visits he must become aware of chemicals that are present in the activities and especially their potential for aggression to the skin. Obtaining data of substances through its technical details is important to know more accurate data. Maintain good links with local services related to worker health.

2.4 Visits to the workplace

Inspection of the workplace can provide us important data about the conditions under which work is performed. Potentially irritating agents or allergens can be identified and how they reach the skin of the worker.

2.5 Information provided by the employer

With the information obtained, the doctor will get significant subsidies that will help you characterize the "causal" in difficult cases.

2.6 Criteria for removal

We suggest the criteria adopted by the AMA - Work-Related Diseases. Procedures Manual for the Health Services 2001.

-Staging, indicators and parameters for removal of the workers of their jobs.

•Grade or Level 1

Signs and symptoms of skin disease are present, even intermittently. There is no restriction or limitation of performance for only a few activities of daily living, although exposure to certain chemicals or physical agents can increase the temporary limitation. No treatment is required or intermittent treatment.

•Grade or Level 2

Signs and symptoms of skin disease are present, even intermittently. There is some limitation of performance for activities of daily living. Intermittent or continuous treatment may be required.

•Grade or Level 3

Signs and symptoms of skin disease are present, even intermittently. **There is limitation of the performance of many activities of daily living.** Intermittent or continuous treatment may be required.

•Degree or Level 4

Signs and symptoms of skin disease are constantly present. There is limitation of the performance of many activities of daily living that may include intermittent confinement within the home or another home. Intermittent or continuous treatment may be required.

•Degree or Level Five

Signs and symptoms of skin disease are constantly present. There is limitation of the performance of most activities of daily living that may include occasional or constant confinement indoors and another domicilio. Tratamento intermittent or constant may be required.

CHAPTER 3

MAIN occupational dermatitis

3.1 irritant contact dermatitis (ICD) ICD - 10 L24

Unlike allergic contact dermatitis, it is not necessary prior sensitization. The pathophysiology of irritant contact dermatitis does not require the involvement of immunological mechanisms. Thus, it can appear on all workers exposed to skin irritants, depending on its concentration and exposure time and frequency of contact with the irritant. Frequent contact with water, soaps and detergents to promote irritation.

The clinical picture varies according to the irritant and may appear in the form of dermatitis indistinguishable of acute allergic contact dermatitis, ulcerations to deep red in chemical burns. The chronic irritant dermatitis is more frequent than acute or accidental. Repeated assaults by low-grade irritant, occur over time. In these cases, skin dryness and cracks are often the first signs that evolve into erythema, scaling, papules, vesicles and gradual thickening of the skin. The irritant contact dermatitis can be readily diagnosed by clinical and occupational histories. Patch testing or patch test are not indicated for diagnosis. But chronic ICDs do not respond well to treatment may take the test to investigate contact sensitization to the drug sometimes used.

3.1.1 Irritant Contact Dermatitis strong (DICF)

•Etiopathogenesis

Strong irritants are chemicals that produce, when in contact with skin, severe inflammatory lesions, the first contact. The severity of injury depends on the toxicity of contact time and concentration of chemical agent. The cement for being abrasive and highly alkaline hygroscopic, produces, under special conditions when contact with skin, shallow and deep ulcerations. The contact time of the mass or cement grout more pressure and friction exerted by the shoe and / or clothing against the tegument are important factors in the onset of these lesions. The fall of cement, cement grout or concrete, or cement dust (ONUB; Essiet, 1986), in quantity, inside the boot or shoe, the more friction and pressure that occur in the area of skin contact with the cement will initially produce intense erythema, ulceration later, ulceration and necrosis in the affected area. Hannuksela (1976) described deep ulcerations in the patellar region, about 12 hours after exposure, in the seven workers who were working on their knees in contact with wet cement. The alkalinity and oxidizing

power of knowledge are important factors in the genesis of these ulcerated lesions. The friction and pressure are factors conditioning, because lesions occur with greater severity on sites where these factors exist, the more accumulation of the mass of cement or concrete.

•Clinical Presentation

Hours after falling mass of cement in their boots or shoes, there is erythema with itching, burning, burning. The next day, it may be remarked in an active phase lesions, ulcerated, or necrotic, depending solely on contact time and alkalinity
cement or concrete.

- Key clinical features of the main contact dermatitis

Irritant contact dermatitis (ICD). Dry skin on contact area. Desquamation with or without erythema. Can evolve with cracks and bleeds. It is important to emphasize that the irritation process will depend on the causative agent (see below the classification of irritants as ICD 10).

Strong contact irritant dermatitis (DIFC). Surge ulceration in the contact area with subsequent necrosis. Stinging, burning pain and symptoms are present. Contact with acids, strong alkalis are the main agents responsible. Another important player is the falling mass of cement or concrete into boots, shoes or gloves. Allergic Contact Dermatitis (ACD) presence of erythema, swelling, blisters and prurido. Ao become chronic, there is a serous crusting sometimes with secondary infection occurs sometimes lichenification (skin thickening). Note: itching, along with other clinical findings, it is a good indicator of Allergic Contact Dermatitis (ACD).

Table 4 - Major irritant contact dermatitis and their respective agents

Irritant contact dermatitis due to detergents (L24.0)

Irritant contact dermatitis due to oils and fats (L24.1)

Irritant contact dermatitis due to solvents: ketones, cyclohexane, chlorine compounds, esters, glycol, hydrocarbons (L24.2)

Irritant contact dermatitis due to cosmetics (L24.3)

Irritant contact dermatitis due to drugs in contact with skin
(L24.4)

Irritant contact dermatitis due to other chemicals: arsenic, beryllium, bromine, chrome, cement, fluorine, phosphorus, pesticides
(L24.5)

Irritant contact dermatitis due to food in contact with the skin

(L24.6)

Irritant contact dermatitis due to plants except food (L24.7)

Irritant contact dermatitis due to other chemicals, dyes (L24.8)

3.2 Allergic contact dermatitis (ACD) ICD - 10 L23

CVD manifest as acute or chronic eczema. In the acute phase, are accompanied often by intense itching, and in the chronic forms of skin thickening (lichenification), peeling and cracking. Classified as allergens, for having presented a positive patch tests, demonstrated approximately five thousand substances (DE GROOT, 1994). Table 5. ACD corresponds to an immunological reaction of type IV. Contacting the substance is able to penetrate the skin and stimulate the body's immune system to produce T cells that release various cytokines,

causing an inflammatory reaction. The DAC results from eczematous skin reaction, immune-mediated T cell with antigen-specific delayed response to an antigen hapten in contact with skin. Move away from contact with the allergen, there may be remission of the picture, but the hypersensitivity

remains latent and reexposições again unleash lo.O incubation period, after the initial exposure, can vary from five to 21 days. In sensitized workers, re-exposed to contact with a sensitizing agent, it is anticipated the appearance of eczema in a period of one to three days and his disappearance from two to three weeks, after termination of exposure.

Table 5 - Major causative agents of Allergic Contact Dermatitis (ACD) ICD - 10

Allergic contact dermatitis due to metals (L23.0)

Allergic contact dermatitis due to adhesives (L23.1)

Allergic contact dermatitis due to cosmetics (Manufacturing / handling) (L23.2)

Allergic contact dermatitis due to drugs in contact with the skin (L23.3)

Allergic contact dermatitis due to dyes (L23.4)

Allergic contact dermatitis due to other chemicals (L23.5)

Allergic contact dermatitis due to food in contact with the skin (manufacturing / handling) (L23.6)

Allergic contact dermatitis due to plants (excluding plants used as food) (L23.7)

Allergic contact dermatitis due to other agents (external cause specified) (L23.8).

Characteristics of chronic conditions:

The chronic conditions are characterized by thickened skin, with cracks, and may exacerbate the reexposições the antigen. The diagnosis and characterization as a work related disease are made based on clinical and occupational history and clinical examination. The identification of allergenic substances (for diagnostic purposes and to prevent new contacts and re-exposure) can be aided by patch tests or patch tests.

3.2.1. Treatment and other approaches

3.2.1.1 Topical

•Astringents

In the stage of vesicles and oozing is indicated for use tampons or immersion of the feet and hands in a normal saline solution, boric acid or potassium permanganate. The lesions are usually dry in three days.

•Emollients

Referred to recover the function of the skin, reduce dryness and itching. Vaseline can be used, cold cream, cream Lanette. Add urea only when the skin is full, otherwise

itching and burning may occur. Targeting warm bath, not with mild soap and loofah.

•Topical Corticosteroids

The choice of concentration, power and vehicle depends on several factors: type, stage and location of the eczema, and the age of the patient.

Creams or lotions: for exudative lesions.

Cream, lotion or gel, or hairy areas for flexor.

Ointment: For dry lesions and the use of adverse crônicas.Efeitos**Corticosteroids: Skin atrophy, telangiectasia, purple streaks.**

If there is recurrence by suspending the treatment, especially when using highly potent steroids and abruptly interrupt their use

- spread or secondary infection: bacterial, fungal;

- perioral dermatitis, acne rosacea - when used on the face;

- sensitization to the actual formula or corticosteroids;

- Systemic use: adrenal axis suppression - cushingoid manifestations.

3.2.1.2 Systemic treatment

If there is secondary infection may be extensive used antibiotics such as erythromycin, cephalosporin, tetracycline, and antifungals: fluconazole, ketoconazole, terbinafine, itraconazole.

- Antihistamines to relieve itching, hydroxyzine, loratadine,

cetirizine.

- Systemic steroids, given in the acute phase of severe exacerbations.

- Treatment with psoralen and UVA (PUVA). This treatment is for patients with chronic, widespread

- Must be performed in specialized centers.

- Cyclosporine: the immunosuppressive action, has dose-dependent toxicity and may be used only under supervision of a physician experienced in the use of this drug.

To summarize:

- Care hygienic places to prevent secondary infection.

- Topical Treatment: steroids and / or antibiotics, emollients, moisturizers.

- Systemic treatment:

- Systemic antihistamines. In more extensive cases, one must employ systemic corticoidoterapia.

- Removal from exposure is essential.

- In case of secondary infection uses: topical antibiotic, or systemic, depending on the extent of the injuries.

Despite the unwieldy, the chronically resulting from occupational eczema respond well to appropriate therapy. If this does not occur, you should check one of the following:

- 1.trabalhador remains in contact with irritants and sensitizers;

2. areas of integument remain due to eczematous excoriation produced by rubbing;

3. self-injury may be occurring (artefacta dermatitis) or the important contribution of emotional factors in the maintenance of acne.

3.3 contact dermatitis with photosensitivity

3.3.1 Dermatitis caused by ultraviolet radiation: ICD - 10 L56

The photodermatoses, also called photodermatitis or lucid, comprise a large number of abnormal reactions of the skin caused by ultraviolet light or visible spectrum of light. Two tables are the most important polar: phototoxicity and photoallergy. Workers in many occupational activities can be exposed by **four or more hours at peak times and be affected if they neglect** adequate protection. Table 6.

The phototoxic reactions (phototoxicity) result from chemically induced reactivity to ultraviolet light and / or radiation, on a non-immune. The phototoxic reactions, by what is known so far, occur within a logic of

dose-response, and the intensity of reaction proportional to the concentration of the chemical and the amount of radiation in certain wavelength.

Table 6 - Occupations most affected by the action of ultraviolet light

- Farmers.
- Fresh produce.
- Fishermen.
- Sailors.
- Gardeners.
- Workers in road maintenance.
- Construction workers.
- Relays.
- Lifeguards.
- Workers in deepwater fields.
- Workers in maintenance services outside.
- Telephone, electricity and others who work in foreign services.
- Buoys-Frias.
- Welders (arc welding).
- With arc welders.
- Operators with ultraviolet germicidal agents.
- Laser ultraviolet and others.

3.3.2 Clinical picture and diagnosis

Phototoxic reactions are manifested by an immediate sensation of burning, redness, swelling, blistering and sometimes blisters. The burning sensation is more pronounced than those observed in ordinary sunburn, but is relieved in the shade. Late erythema and edema may appear after a few hours to one to two days after exposure. In more severe cases blisters may appear.

A localized hyperpigmentation can be observed after the reaction, and in some cases it may be the only manifestation. The intensity of the disease depend on the amount of radiation, skin type, the site of exposure and concentration of the substance. Lesions of phototoxic reactions are confined to areas of skin exposed to light, typically in one or more areas of the face, tips of ears, the "V" neckline, neck, back of the neck, extensor surfaces of forearms and back of hands. The presence in other areas will depend on the worker's clothing. Photoallergic reactions are usually characterized by eczematous lesions, occurring in erythema, edema, infiltration, and vesiculation in

more severe cases, blisters. The lesions may extend beyond the

exposed areas, aggravating the areas previously observed cobertas. Pode be a mild widespread dermatitis. To the extent that reduces dermatitis, pigmentary changes and skin thickening may become prominent.

Some patients react to extremely small amounts of light energy. The impairment charge of photoallergy wave lie in the range of long-wave ultraviolet (UVA). A serious complication of photoallergy is the development of a persistent reaction to light. The disease is characterized by extreme photosensitivity that persists despite the removal of all contact with the photoallergen. You may get an extension of the action spectrum of light, which causes small exposures to UV radiation trigger photosensitivity.

Photoallergic reactions (photoallergy) are differentiated from phototoxic reactions the nature of immune response that occurs only in individuals who were previously sensitized by simultaneous exposure to photosensitizing substances and radiation properly. The photoallergy seems to involve biological processes similar to those of allergic contact dermatitis, except by ultraviolet radiation, the conversion of the hapten in complete allergen.

The diagnosis of photodermatoses often suggested for the distribution and character of skin lesions. The frames require photoallergy for confirmation, a more thorough investigation that includes photopatch test (Phototest), which must be performed by a specialist (dermatologist) familiar with the technique.

Table 7 - fotossensibilidade reactions caused by various agents

1) For systemic action:

- a) antiarrhythmic agents, amiodarone, methyldopa, propranolol, quinidine;
- b) antibiotics: tetracycline, dimetilclortetraciclina, nalidixic acid;
- c) oral antidiabetic sulfonamide;
- d) non-steroidal anti-inflammatory, piroxicam, benoxiprofen, acetylsalicylic acid (ASA), phenylbutazone and oxifenilbutazona, ibuprofen;
- e) antineoplastic agents (methotrexate, vinblastine, 5.fluorouracil);
- f) derivatives of quinine - chloroquine;
- g) diuretics - thiazide, chlorothiazide, furosemide;
- h) retinoids, isotretinoin, etretinate.

2) For topical:

non-occupational causes.

3.4.3 Clinical picture and diagnosis

The ulcers caused by exposure to chromium develop, usually in damp areas, such as nasal mucosa, or skin areas in which there were previous injuries such as abrasion or break in continuity due to injuries. Ulcers may appear on the junction of the phalanges of the finger (or outer surface of extension), the most salient points in or near the fingernails, among other locations.

Have from 2 to 4mm in diameter, with raised borders and clearly marked with the bottom excavated. They are very sensitive and painful and may be covered by a crust. A secondary bacterial infection is common. The evolution is slow and can leave scarring. The continuity of exposure can lead to the formation of a halo around the necrotic ulcer with growing size. In the process of chroming exposure to chromic acid mist can cause severe damage to workers' health.

Table 8 - Shares of hexavalent chromium on the integument, mucosae and upper

Periungual lesions.

Ulcers in previously injured in the integument.

Ulceration and perforation of the nasal septum.

Brown tongue and teeth.

Rhinitis and asthma attacks.

Cancer of the bronchi (bronchogenic carcinoma).

Other irritants produce ulceration without distinguishing clinical features the diagnosis is made based on clinical characteristics and history of exposure to chromium or other irritating agent. When the causative agent is chrome, should be investigated other harmful effects such as ulcers, nasal septum perforation, cancer of the nasal septum and chronic effects on the lung.

3.4.4 Treatment and other behaviors

According recommends Ali (1995), the treatment of ulcers caused by chrome should include:

a) the cessation of exposure to harmful agents;

b) cleaning of ulceration using saline 0.9% or a

ascorbic acid solution prepared by dissolving a tablet of 1 g of effervescent vitamin C in 10ml of distilled water or saline. The solution should be kept in dark bottle and renewed semanalmente. Deve to make a dressing with cotton wool soaked in the solution, leaving about an hour and afterwards, use a healer. Repeat the procedure for five days, keeping

the healer to cure.

In cases of irritation or nasal septum perforation, using cotton soaked in this solution for about an hour with the patient lying for five days. Use saline nasal use several times a day during the treatment. The return to the same desktop, after healing of ulceration or perforation of the septum, can cause recurrence.

3.5 contact urticaria ICD - 10 L50.6

Urticaria is characterized by the eruption of wheals, wheals that will outline irregular and short duration and usually pruritic. The papules may coalesce to form large plaques. The injury is an allergic reaction that occurs as a result of histamine release from mast cells located around the dermal vessels in response to contact with a chemical or physical. Contact urticaria is the term used generically to describe the acne caused by non-traumatic and that develops these by direct contact with intact skin, may be allergic or not. A rash or allergic contact hypersensitivity is a picture of individual and its prevalence is difficult to determine. Urticaria caused by heat is very rare. The identification of the causative agent can be extremely difficult, especially in chronic cases in which 70% are of obscure origin and may be due to occupational exposure. Urticaria-related work. The work can act as a necessary cause, in normal workers, or act comodesencadeador or worse, in workers hypersensitive or allergic the same chemical or physical agents.

3.5.1 Clinical picture and diagnosis

Hives can vary from millimeters to centimeters or form larger plaques. There may be a washout in the central lesions and formation of circular shape, serpentine or arcade. There is a severe form called angioedema or edema or Quincke giant urticaria, affecting most frequently the eyelids, lips, tongue and larynx, which can be lethal if there is edema, not treated early. Urticaria due to heat and cold is characterized by the appearance of wheals within minutes of direct application of heat or hot object or environmental exposure to cold.

The papular appearance, the rash of injuries and short duration make it easy to define the diagnosis of urticaria. Cases of urticaria caused by heat and cold can be confirmed by placing a test tube with hot water (from 38 to 42) or ice, respectively, on the skin, the wheals appear within minutes.

3.5.2 Treatment and other behaviors

The therapy depends on the severity. Some cases can be controlled through the use of antihistamines. In others there is need to involve steroids. In severe cases that occur with swelling of the larynx and glottis, bronchospasm, nausea, vomiting and hypotension is indicated the administration of epinephrine subcutaneously or intravenously.

3.5.3 Prevention

The prevention of work-related urticaria is based on monitoring of environments, working conditions and the effects or health hazards, as described in the introduction to this chapter. The environmental risk factors involved in determining the disease can reduce their incidence among occupational groups at risk through the elimination or reduction of exposure or control, as in cases secondary to exposure to heat and cold. For some groups of workers may be advisable to use insect repellent creams.

The handling, preparation and application of pesticides shall be made by trained personnel observing safety standards, special care with the application equipment and the use of protective gear. You should try to substitute other products with less toxicity.

Production, transportation, use, trade, application and disposal of packaging (toxic waste) pesticides must comply with the standards established by Federal Law No. 7.802/89 and regulations specific to states and municipalities. Also observe the provisions of the NRR, the Ordinance / No 3.067/1988 MTE.

Table 9 - Major types of hives classified by ICD - 10

Allergic urticaria (L50.0) Occupational exposure to pesticides and other specific chemicals. Urticaria due to cold and heat (L50.2) Occupational exposure to cold and heat.

Contact urticaria (L50.6) Occupational exposure to chemical, physical and biological agents, specific, affecting the skin.

3.6 acneiform eruptions

3.6.1 Elaioidermatitis or follicular ICD - 10 L72.8

The elaioidermatitis follicular or follicular dermatitis or acne or folliculitis for heavy oils from petroleum or mineral oils are acneiform eruptions and presenting as follicular papules and pustules that occur in areas of exposure in susceptible workers, such as the forearms and thighs. The mechanism of action of cutting oils and other fats, begins by irritation of the follicular, followed by the same obstruction. The same agents (oils and fats, minerals) can cause other conditions such as irritant contact dermatitis and allergic. The classic descriptions of acne by oils and

greases are for workers in machine shops to repair cars and other vehicles and the metallurgical industry, using cutting oils. With the diffusion and adoption of care for personal hygiene and cleanliness of the clothes, the disease incidence has declined.

3.6.2 Clinical picture and diagnosis

The oils and fats by acne is characterized by comedones and follicular papules and pustules, usually located in the hands and forearms may extend to the abdominal area, thighs and other areas covered, if the clothes in contact with the skin is dirty oil. The presence of black spots on the follicular ostia suggest the diagnosis. Deep. Superficial injuries involve the epidermis, also known as follicular elaiocniose. The lesions involve the deep dermis and subcutaneous tissue, and occupational called furunculosis. Table 3. Can occur in three clinical elaiocniose: shape papular, pustular form and mixed form.

The diagnosis is based on morphology, location of lesions and history of occupational exposure to oils and greases of mineral or synthetic.

Table 10 - Etiopathogenesis of lesions produced by oils, greases and oily dirt on the skin

- Irritating effect of cutting fluid on follicular bone.
- Mechanical obstruction - most cutting fluid particulate material promotes bone follicular obstruction and facilitates bacterial infection.
- Penetration of the cutting fluid through the duct hairy irritating him and making it easier to bacterial infection.

Table 3 - Clinical acneiform eruptions by cutting fluids

Folliculitis - Action irritative perifollicular

Papular Elaiocniose - perifollicular erythematous papules, comedones black

Elaiocniose papulopustular - Presence of erythematous papules, pustules and papulopustular

Furunculosis - Infection of bone level in follicular dermal irritation caused by cutting fluids, grease and dirt

3.7 dyschromias

Definition: it's all change in color of skin, mucous membranes and attachments, subject, maintained or worsened by stakeholders in the occupational activity. Depending on the agent, there can be changes in pigmentation that can be higher - hyperpigmentation - and for less - hypochromia. When the exposure determines hypochromia in some areas

and accumulation of pigment in others, we call this picture of pigmentary

3.7.1 Melanoderma ICD - 10 L81.4

Melanosis melanosis or hyperpigmentation of the skin is due to increased melanin. The pathology of the work are acquired melanosis caused by exposure to chemical agents of occupational origin. The melanosis melanosis or occupational in nature can be caused by physical agents, including repeated trauma, friction, thermal burns, artificial and natural ultraviolet light due to sun exposure, and chemicals such as petroleum hydrocarbons, tar, coal, asphalt, bitumen, paraffin, tar, coal tar, creosote, tar, cutting oils, anthracene and dibenzoanthracene, among others. Certain wood dust can also cause melanosis.

It is important to remember that these agents can also produce other skin effects, as photodermatoses, folliculitis, acne and epithelial hyperplasia. Arsenic and its compounds, chlorobenzene and dichlorobenzene, bismuth, cytostatics, nitrogen compounds, dinitrophenol, Naphthols added to dyes, para-phenylenediamine and its derivatives, quinine and derivatives, salts of gold and silver can also cause melanosis.

3.7.2 Clinical picture and diagnosis

Hyperpigmentation occurs in the areas of contact with the agent in form of stains clearly showing pigmentary changes in the contact region. In general, the areas most affected are the face and neck and the trunk is less affected. Can be found, often, the scalp lesions, with erythema, itching and flaking. The histological picture shows focal increase of melanin pigment in the basal layer of epidermis, with perivascular lymphocytic infiltrate adnexal and discreet. Edema can be observed and chromatoforese. O differential diagnosis must be made with other causes of melanosis acquired, systemic diseases and endocrine-metabolic and infectious with melanoma, where located.

The improvement of symptoms occurs with the elimination of exposure to the causative agent. In some cases, there is leakage of melanin to the epidermis, pigment incontinence, which may cause permanent hyperpigmentation.

3.7.3 Occupational leukoderma (vitiligo includes occupational)

ICD - 10 - I81.5

Leukoderma leukoderma means or hypopigmentation of the skin. The Occupational leukoderma may be caused by physical and chemical agents. Among the physical agents are thermal burns, ionizing radiation (radiodermatitis or necrosis induced by x-rays) and the repeated trauma to the skin, which can lead to hypo-or depigmentation. Among the chemical

agents stand out alkylphenols (phenols and catechols), which can irritate or depigmented skin areas directly exposed the monobenzyl ether of hydroquinone (Mbeh-antioxidant) used in synthetic rubber industry, and hydroquinone (HQ), used industry in paints, plastics and insecticides. Cases have been described in workers exposed to other alkylphenols, such as para-tertiary-butyl phenol (TBP), the for-terciárioaminofenol (TBA) and arsenic compounds. The agents that cause irritant contact dermatitis or allergic may induce leukoderma temporary or long term. Vitiligo affects about 1% of the general population and in about 30% of cases there is a familiar occurrence. Proven cases of occupational leukoderma are relatively rare but can occur epidemically in certain groups of workers exposed.

3.7.3.1 Clinical symptoms and diagnosis

Clinically, the chemically induced leukoderma is indistinguishable from vitiligo. Generally, hands, wrists and forearms, face, eyelids are most affected. Regions for and may have symmetry between the lesions. The depigmentation may also appear in the armpits, genitals, and shoulders. No changes are described pigmentation of head hair and eye color. Often the simultaneous presence of dermatitis is noted. An occupational etiology is defined by a history of exposure to agents and producers of leukoderma factors and the observation of the activities developed by the worker, for example, how you use your hands at work and the presence of more cases or outbreaks in the same section

or workplace. The patch test or contact test may indicate allergic hypersensitivity, the simultaneous action of lightener.

3.7.3.2 Treatment and other behaviors

The cessation of exposure to the etiologic agent is mandatory. The use of photoprotectors is indicated, since the burn is easily achieved by sun exposure. Some agents destroy the melanocytes, as monobenzyl ether of hydroquinone, and in these cases, leukoderma may be final. The treatment follows the same scheme one used to treat idiopathic vitiligo

Nail dystrophy - onychopathies

They are nail disorders and their various components are manufactured, maintained or exacerbated by biological, chemical and physical environment work. present in your daily activity, the employee

may suffer various beatings on fingernails. The lesions are extraordinarily diverse and extensive range of feature changes that occur on the surface, extent, thickness, consistency, adhesion, color and shape of the blades nail. **The nail plate is formed by keratin produced by the cells doleito nail. This is derived from an invagination of the epidermis and is the most extensive training of the human nail keratin and tissues that comprise the unit are ungueal.**Exercem protective functions and aesthetics, thus contributing effectively to the functionality of the fingers. In occupational therapy, **trabalhadorestá** in frequent contact with different actors, which in determinadassituações, may affect the nails. However, much poucotem been described on these agressões. **Agentes biological, chemical and physical harm the nail bed may compromise the functionality and the fingers of their hands, reducing the efficiency and productivity.**

3.8.1 onychopathies caused by biological agents

Fungi, yeasts, bacteria and viruses are the main biological agents that compromise the nail plate.

3.8.2 onychopathies caused by chemical agents

They can reach the nail plates, promoting its partial or total destruction. Acids, alkalis, solvents, resins and other chemicals, potentially irritating or sensitizing may lead to irreversible damage in transient and nail plate.

3.8.3 onychopathies caused by physical agents

In the occupational, physical agents are the largest producers of onychopathies. Trauma, friction, pressure, heat, cold, humidity, ionizing radiation, microwave and vibration are important factors in compromising the skin and its annexes.

•Heat

Burns in February. Degree, there may be destruction and alteration of the matrix, nail dystrophy and appearance of onychogryphosis. Burns 1. Prominence degree can promote lateral or distal hit the nail, making it brittle and cracked on the edge terminal.

•Cold

Prolonged exposure to cold and normal can affect the nail matrix, which can be altered in oncogenesis with deep transverse grooves or Beau's lines. These problems may be exacerbated in susceptible workers, patients with erythema pernio or Raynaud's phenomenon.

•Humidity

Workers exposed without adequate protection in the feet or hands, may suffer because of the moisture soaking. In the plantar or palmar

tissue may occur so-called immersion foot and hand immersion subungual tissue involvement and detachment of the nails (onycholysis). Accordingly, the nails are subjected to secondary infection by fungi, yeasts and bacteria.

•Prevention

1. improvement in the quality of personal protective equipment, protecting against moisture and excessive sweating;
2. rest period and turnover in work in progress;
3. detecção and treatment of workers affected.

3.9 Occupational Skin cancer

The exposure of workers unprotected or poorly protected, solar radiation, is now the leading cause of occupational skin cancer. Other nations chemicals when in contact with normal skin can also cause skin cancers. Among the most important are: creosote pixe, arsenic, waste oils, used grease, Agent **presence of some chemicals with polycyclic aromatic hydrocarbons** and others. Table 4.

The highest incidence of skin tumors among workers in fair skin (Caucasian) exposed to sunlight is actually verified. The incidence of basal and squamous cell epithelioma is more frequent in these workers.

The state of Arizona has a high incidence of exposure to solar radiation. A recent study conducted in this state in over a hundred thousand people, showed that the incidence of skin cancer is around 270/100.000 in both sexes and Caucasian present rates

10 times higher than Hispanics (Harris, 2001). Basal cell carcinoma is the type that occurs most frequently, about 75%, however, melanoma, in 1935, had 1/1500th the incidence reached in 2000, the mark of 1 / 74 (Lim et al, 2001) The location, 65% of squamous cell carcinomas were located on the face and neck, arms 20%, 14% of women in the lower limbs

and 3% in the lower limbs of men. Skin cancer by other sources of UV. Chronic exposure to electric welding in the various types used in the welding industry, and without proper protective skin can cause rashes in areas exposed repeated

and that, over time, may determine the onset of skin cancer in these areas, especially the basal cell type. It is believed also to occur a higher incidence of melanomas in fair-skinned workers exposed to sunlight (ZHAO, 1998; WANG; Setlow, BERWICK, 2001).

3.9.1 Prevention

Important factors: avoid exposure during peak hours, ie between

10h and 15h. Use suitable protective for the time that will be exposed to and the color of their skin. It is very important to use sunscreen properly, it is recommended to reapply it 15 to 30 minutes after exposure. The sunscreen is a misperception of the first ways used to prevent and its improper use can lead to false protective sensation. Studies in this direction show that the sunscreen did not protect the user in 55% of the time (Wright, M; WRIGHT, S., Wagner, 2001). In outside work, if it occurs profuse sweating, it becomes necessary to further application in areas exposed to light two to three hours after the first application (Diffey, 2001).

Adequate protection is the proper use of glasses, hat, cap with flaps and clothing with long sleeves. Garments with a capacity retention of UV light are being marketed in some countries and its improvement will contribute to better protection of the worker. Cotton and viscose are tested in vitro by spectrophotometry, and in vivo through the minimum erythematous dose (MED) with the tissue and without the fabric. The results showed that it can achieve good levels of protection with the use of this type of clothing (HOFFMANN, 2000). The color of the clothing and the use of detergents and triazine derivatives, which absorb UV radiation, increased the capacity of protection in more than four times (Steven, 2001). Wear sunglasses when there is a need for appropriate long-term exposure in environments with strong UV light emission and / or UVB, in some work situations, and depending on the type of skin color, use of sunscreens may be helpful.

Manufacturers of protective clothing against the emission of UVA and UVB light show the following trend classifying the protection offered by different tissues in three categories:

Good protection UPF 15 to 24
Very good UPF 25-39
Excellent UPF 40-50

(Note: unit = UPF protection factor **The protection factor UPF measures the protection the clothing with tissue** specifically gives the skin. For example: clothing with an SPF of 50, this means that only 1 / 50 of UV light can penetrate the tissue, ie, only 2% of the rays pass through the tissue affecting the skin. Manufacturers work with woven UPF factor which varies from 15 to 50. Depending on the type of activity, we chose the factor that the worker must use. If exposure is 8 hours / day factor should I choose 50 that will give 98% protection, if the exposure is less intense, we can use factor 15 that will give 93% protection against UVA and UVB. Important as it ages and gets the clothes washings, their power

of protection diminishes. The washing of these garments with optical brighteners and chemical agents can maintain their appropriate protective ability

4 PREVENTION OF Occupational dermatitis

4.1 Concept

Means assessing the work environment to meet potential and actual risks to workers and propose measures to counteract these risks (DIAS, 1999, Oliveira 1999).

The knowledge of the real risk and potential risk involves efforts aimed at neutralizing its effects. Risks should be assessed according to the activity performed because we know the function of each worker in the activity may require different processes of prevention. Schematically, we can act on three different levels to protect the worker.

4.2 Primary prevention: health promotion

Desktop - the buildings and the various industries and industrial plants must comply with the rules which comfort, welfare and safety.

•Health structure for easy access and allows good personal hygiene.

•Restaurant with food appropriate to the climate and the activity performed.

•Training center.

•Guidance on specific risks relating to the activity.

Safe working methods.

•Guidance on general diseases: tuberculosis, AIDS, diabetes, hypertension, stress and others.

•Social ills of smoking, alcoholism, drugs, medications, anxiolytics psychotropic others.

•Standards of hygiene and immunization.

4.3 Secondary prevention

At this level we operate by detecting potential injuries that are occurring with the worker.

•Through the outpatient clinic of the company.

•Through regular inspection to workplaces.

•Through regular examinations and early treatment.

Here we can act immediately, neutralizing or minimizing risks, and prevent the acne to install and hit the exposed workers.

4.4 Tertiary prevention

At this level, the employee presents chronicle it shows injuries or chronic phase or is found sensitized to some agent present in the

workplace. In this case, it is essential to adopt appropriate therapeutic measures such as withdrawal from the desktop, patch tests in order to detect the presence of possible allergens. In the specific case of allergy to cement (chromate and cobalt), there will be unable to return to the same activity, in which case the employee must be restored to another kind of activity which can act away from risk. Flowchart 1.

4.5 Prevention: concept

Recognition of activities and workplaces where there are chemicals or physical agents and biological risk factors arising from work organization, potentially causing disease.

- Identification of problems or potential harm to health arising from exposure to risk factors identified.

- Proposing measures to be taken for disposal or reducing exposure to risk factors and the promotion and protection of workers' health .- Guidance and information to workers and employers.

- Upon confirmation of diagnosis or even suspicion of the disease and its relationship with work, health services responsible for the care workers must implement the following actions:

- Assessing the need for removal (temporary or permanent) worker exposure, the sector of employment or work as a whole. This procedure may be necessary even before the confirmation of diagnosis, before a strong suspicion.

- If the worker is insured by the SAT of Social Security, ask the company to issue the CAT, the LEM fill and send to the INSS. In case of refusal to issue the CAT by the company, the doctor should do it.

- Monitoring and recording of progress, particularly if there is clinical worsening with the return to work.

- Notification of grievance to the Information System Morbidity of SUS, the DRT / MTE and the union.

- Shares of epidemiological surveillance aimed at identification of other cases through active search in the same company or workplace or other companies of same industry in the geographic area ca.

- If necessary, make the identification of the offending agent (physical, chemical or biological) and working conditions determine the disorder and other contributing risk factors.

- Inspection of the company or work environment where the patient and other companies in the same industry in the geographic area ca, seeking to identify which risk factors for health, measures

collective protection and PPE used. It may be important to verification of the existence and adequacy ofPPRA (NR 9) and PCMSO (NR 7), the Ordinance / MTE

No 3.214/1978.

- Recommendation on the protection measures to be adopted by the employer, informing the workers. Health protection and prevention of exposure to risk factors involve engineering measures and industrial hygiene, changes in organization and management of labor and control of medical

exposed workers, including:

– Replacement of agent, substance, technology or tool for working with safer, less toxic or harmful (fluxograma 2);

- Isolation of the machine, potentially harmful substance or agent, by enclosing the process by eliminating or reducing exposure;

- Hygiene and safety, such as deployment and maintenance of local exhaust ventilation adequate and efficient, fume exhaust, control of leaks and incidents through preventive and corrective maintenance of machinery and equipment, and systematic monitoring of aggressors;

– Adoption of work systems and secure operating through the classification and labeling of chemical substances toxicological and toxicity;

– Reduction of exposure time and the number of exposed workers;

– Information and communication of risk to employees;

- Use of EPI, especially glasses and masks appropriate to each type of exposure, so additional measures for collective protection.

OBJECTIVE 5

The main purpose of this Manual is to provide professional SUS material and grants for the care of patients seeking the service and who are suffering from various skin disorders, some allegedly linked to labor activity. Establish standards of care, using protocols previously developed in order to standardize the collection of data on skin diseases resulting from occupational

5.1 Benefits

The use of protocols and manuals has a number of advantages for the NHS and for the worker. We highlight:

•The patient chart pattern that can be used nationwide.

•Ease in completing and obtaining the basic data on the worker at the entrance of the service.

•Ease in directing the worker with occupational disease to the care system, with the connector itself for each disease mentioned.

•Easy to computerize the entire outpatient procedure nationally.

•Once computerized, the system can provide more precise data for occurrences of occupational diseases and

its prevalence throughout the country

- The cost reduction can be measured by the ease and speed in obtaining and entering data specific and overall, making it easier to establish the prevalence of diseases and injuries suffered by the worker, both of formality and informality.

.6 EPIDEMIOLOGY

The changes in the mucous membranes and skin and appendages of the employees are experienced in many activities and represent significant portion of occupational diseases. They do not get the statistics and even the knowledge of experts. Many are self-treatment, others are treated in the ambulatory of the company. Some come to the clinician and the specialist doctors in the consortia that provide assistance under an agreement with the INSS. The true incidence of dermatoses in the working class is unknown. Statistics published by government agencies represent only part of workers who work in formality and were somehow served by social security services. A good portion of the workers have no formal or CAT issued by acne present with little or no reduction reduction of working capacity or afraid because of the resignation conceal your acne. The available statistics most likely represent the tip of iceberg a major cause of illness among workers who are the injuries sustained in the skin resulting from occupational activity. The collection of data that the National Network for Health Care Workers (RENAST) will generate, may be an important tool for better understanding of much of the reality of the occupational health of the working class. In developed countries, estimated that about 1% of active workers may have occupational dermatosis. For these general aspects that cause the illness are met workers from different industries and functions. Due to its high prevalence, occupational dermatitis have received attention from the state definitions of care policies, especially those sickened, with the creation of Technical Standards of Social Security, Norm of the Ministry of Labor and Employment, Diagnosis and Treatment Protocol by the Ministry of Health

METHODOLOGY

Models were used schedules established services like Sesi

- Division of Industrial Medicine - Catumbi and Vila Leopoldina - and in the Department of Occupational Dermatology Fundacentro, copies of records of other works were modified according to the needs of the service. These models were reduced and simplified in order to provide the doctor who will make the first visit a plug at the lowest possible number

of basic data. The use of this material may result in a form that can be adapted for computerization, as soon as possible. Number of documents-based: we use the database that we have in our computer system and consultations with several specialized books and review the topic. Methods used to ensure quality and applicability of evidence and sources, using sources recognized by scholars of the subject and service experience in the field of Occupational Dermatology. Methods used to formulate the recommendations: literature review and service experience. Cost analysis: every action in the area of occupational health should be focused on the preventive aspects, preferably for implementation of collective measures of protection and ultimately to the individual measures. The social cost of a worker carrying occupational dermatosis is high when considering the costs of absenteeism, medication change of occupation and vocational rehabilitation. Method to validate the protocol: pre-test in the training and care services for training of trainers.

RECOMMENDATIONS

8.1 Epidemiological surveillance

Suggested Criteria for Sanitary Vigilance to undertake research on occupational dermatitis in business:

a) Frequency of clinical cases of skin diseases that come to the health network, the same company or similar companies.

b) the occurrence of more than one case per month of occupational dermatosis in the same company.

c) the severity of injuries - injuries that compromise over 20% of body area.

d) When there is nasal septum perforation resulting from occupational activity in electrodeposition of metals in the same company.

e) When workers (minors) seek the service to meet any type of skin injury resulting from occupational exposure.

Note: chemical burns to a large extent the body, are dermatoses involving emergency.

When care is not feasible, refer to center of greater complexity.

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ANNEXES

Annex A - Model of care - Version III - December 2005

1. Patient identification

Name: Sex: (m) (f) Age: Weight: Alt:

2. Forwarded by

(1) union (2) Covenant (3) Medical Service Company (4) Convention (5) Service PCMSO (6) Center of Expertise (7) Cerest (8) Others.

3. Seen previously pursued

Time: Days () Month () Year ()

4. Current Activity

Time: Days () Month () Year ()

5. Occupational anamnesis

Personal history of atopy (1) asthma (2) allergic rhinitis (3) atopic dermatitis

Family history of atopy (1) asthma (2) allergic rhinitis (3) dermatitis atopic

Previous skin disease Yes () No () (window may open with ICD-10)

He was relieved Yes () No () Days () Months ()

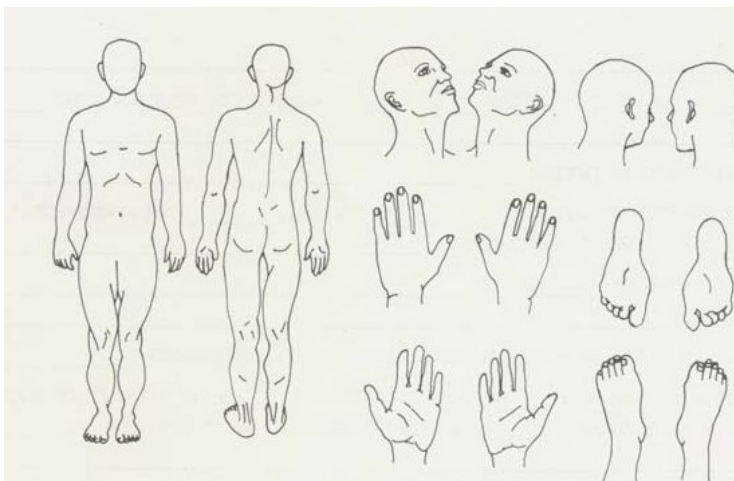
Occupational Yes () No () Non-occupational Yes () No ()

6. Physical examination

Description and location of injury:
Check the affected areas on the doll.

There is symmetry? Yes () No () Color Form

There is impotence of the affected area? () Yes () No



7.Diagnosis, ICD-10

8.Differential diagnosis; ICD -10

9.Complementary tests: patch tests, histopathology, and others.

10. Proposed treatment:

11. Inspection of the workplace. While it is useful and better knowledge of agents and environment involved in the pathogenesis of acne.

12. Information provided by the employer. Get the information Defonte, the most reliable possible. Check other sources when the data are conflicting.

The Internet is an important tool today, where many good information can be obtained. Establish a causal connection. Occupational or nonoccupational.

Justify.

Treatment: Ensure that the network will offer appropriate drugs for treatment of skin diseases.

Appendix B - Occupational Dermatoses

Dermatological exam

Systematization: examination of the skin and its appendages (mucosa, hair and nails).

Getting Started:

Recognition of elementary lesions - NOTE: This protocol presents images of various elementary lesions.

To look for? Dermatoses more frequent in the general population. Mycoses, rash, changes in texture and skin color.

Preexisting skin.

Atopic dermatitis, xerosis, ichthyosis vulgaris, lichen planus and psoriasis.

Dermatoses more frequent in Occupational area.

Construction, metal electroplating, metal industry, artifacts rubber, fiberglass.

Anamnesis - patient data.

- Complaint and duration.

- Personal and Family (APF). There are families with a history skin diseases, allergies, asthma, rhinitis, reactions to food, medicines. If there is mention which. Emphasize that family had a history of allergy.

- Systematization of the skin examination.

- Conditions for a good dermatological examination:

Good lighting - natural or artificial.

Daylight is best.

Good magnifier is useful to see details of certain colors or injuries.

UV lamp or Wood is useful in certain dermatoses such as erythrasma tinea capitis, pseudomonas infections, scabies, vitiligo, porphyria and other changes in pigmentation.

- Examination of the patient: using a well lit with natural light or fluorescent.

The light should come in behind the examiner.

- Examination of the lesion: their form, location, distribution.

If the lesions affecting large areas of skin, examine them from a

Tratado de Toxicologia Ocupacional

distance of two to three feet, approaching gradually to about 20cm.

If necessary, use magnifying glass to see details about lesão. Obter: initial location of the lesion or lesions, distribution, evolution, or by continuing outbreaks. Itching, pain, burning, burning, intensity and duration.

- Treatments used: the one you used, there was an improvement, remained stable, worsening.

There are factors that aggravate or irritate?

The lesions have some correlation with work, leisure, work at home, food, medicines, others.

Check if there are mucosal lesions, skin annexes, search linfonodos. Important to examine the nasal cavity and oral cavity, as some occupational activities may have important lesions in these locations.

Identification of elementary lesions:

- Changes in color and thickness of the skin.
- Lesions with liquid content.

Solid lesions.

- Losses tissue.

- Changes in skin color:

vascular blood stains for vascular constriction or dilation.

Erythema, pallor, angiomatous stain, stain anemia, purpura, telangiectases.

Dark spots increased or decreased by melanin.

Pigmented spot: ephelides = mackerel

Figure 1: Stain pigment: ephelides = sarda. Foto: Salim Amed Ali



Suelen Queiroz

Keratosis = hyperkeratosis. Increased stratum corneum: follicular hyperkeratosis.

Figure 2: Changes in the thickness of pele.Foto: Salim Amed Ali



Lichenification - increased thickness of the epidermis with accentuation of grooves and color.

Figure 3: Calodidade.Foto: Salim Amed Ali



Edema - Thickness increase the accumulation of fluid in the dermis or hypodermis.



Figure 5: allergic contact dermatitis with erythema, edema, vesicles and exudation serosa.foto: Salim Amed Ali

-Infiltration: presence of cellular infiltrate in the dermis. By vitro pressure results lesion color latte.

-**Sclerosis:** hardened skin, leathery, slightly depressing. Results of the amendment

Of collagen fibers.

-**Atrophy:** thin folds due to decreased tissue with reduced thickness. Atrophy is known as linear vibice.

Keloid: Results of fibrous hyperplasia, consistent with barely edges defined in the dermis that occurs frequently after surgery, trauma or burns.



Figure 6: Scar: Results of the destructive process of the skin. Ulcer produced by chromium hevalente next scar ulcer crômica.Foto: Salim Amed Ali



Figure 7: scar queloideana.Foto: Salim Amed Ali

. Figure 8.Vesículas - Allergic contact dermatitis disidrosiforme by



cutting oils. Lesions containing liquid

Figure 9: Photo: Salim Amed Ali (Figure 11) Whelk - lesion containing



pus diâmetro to 1cm.



Figure 10: Hematoma - Collection of blood in the skin or subcutaneous.
Photo: Salim Amed Ali

Acneiform lesions with papules and pustules on workers with dermatitis by oils and greases; this clinical picture is known as "elaiioconiose.

Abscess - collection of pus in skin or subcutaneous. There may be heat, pain, fluctuation.



Figure 11: ulcer.



Solid lesions.

Result from inflammatory or neoplastic processes. Can achieve together the epidermis, dermis and hypodermis. Includes: papule, papular plaque, tumor, vegetation, nodule, urticaria.

Papular lesions due to obstruction by the sebaceous gland oils and / or grease

Papular plaque: Lesion may be individual or group of papules.

Node: solid lesion localized, high or not, 1-3 cm in diameter.

Tumor: solid lesion, high or greater than 3 cm diameter. Note:

Word usually used for neoplastic process.

Wart mole: Acrodordon.

Vegetation: pedunculated papule sometimes looks like cauliflower, bleed easily.

Verrucose: lesion of the hard surface in the form of papular papule or plaque.

Urtica: elevated lesion, color rosy red, itchy and of short duration. Results of acute exudation of the dermis.

Tissue loss: Result of the disposal or destruction of skin tissue.

Scale: epidermal sheets are eliminated spontaneously or due to the action of physical or chemical agents.

Erosion or ulceration: loss that affects the skin surface.

Excoriation: loss of superficial skin by mechanical action.

Ulcer - Ulceration which can become chronic and when leaves persistent scar heals. In the photo below ulcer produced by contact with copper salts in electrodeposition of metals.

Cleft or rhagades - Linear loss of epidermis and dermis.

Fistula - Channel orifice in the skin, draining deep focus of suppuration

Or necrosis.

Eschar - Loss of tissue due to necrotic process.

Crust - concretion

Formed in an area of tissue loss. It may be serous, purulent or bloody. (Clinical picture of allergic contact dermatitis to cutting oil)

Skin and appendages:

The skin consists of three major layers: epidermis, dermis and

hypodermis or subcutaneous tissue. The hypodermis consists mostly of fat cells and its main function is the thermal insulation and protection against external pressures.

- Attachments skin

Apocrine sweat glands: present in the axillae, anogenital region, perimamilar, external auditory canal, eyelids. In the breast region is modified remnants of the gland. Eccrine sweat glands, are distributed throughout the integument mainly armpit and palmoplantar. Main function: term regulation and formation of the lipid mantle. Pila sebaceous apparatus, consisting of glands producing sebum (fat) are distributed throughout the integument, except palmar plantar region. Its size is generally inversely with the length of hair to which they are linked, being highly developed in the nasal and face where the hair is usually undeveloped.

- **Nails:** keratinized structures are composed of three parts:

Root, nail bed and freeboard.

Presence of muscle: striated and smooth muscles.

Smooth-muscles of the skin, mammary areola, hair erector, dartos The scrotum.

Striated, are found in the skin of the neck and face (mime).

- Innervation of the integumentary system.

Motor innervation.

Adrenergic: function: vasoconstriction, contraction of the arrector pili muscle,

Eccrine and apocrine secretion secretion palmar plantar (linked to stress).

Cholinergic: function: eccrine secretion widespread in temperature regulation. Sensory innervation: functions: temperature, itching and pain through nerve endings, tactile function by means of Meissner corpuscles; pressure through the corpuscles Paccini.

- **Functions of the skin:** physical protection and immunology, perception, secretion,

Term regulation.

Lipid mantle is formed from the secretion of sweat and sebaceous glands, acts as regulator of the pH of the skin, regulates fungal and bacterial flora present in the epidermis.

Techniques of dermatological palpation.

Digit pressure: erythema and edema allows distinguish from other lesions.

Digital clamping: check consistency and changes in skin thickness.

Linear compression: Research dermatographism.

Exerts pressure is linear in the back of the patient with suspected Bic pen lid or spherical object with blunt tip, and looks to be about 30s. Erythematous papular linear response is considered positive. Positive result can be symptomatic or asymptomatic. Symptomatic: patients report itching at the site where pressure was exerted. If respondent will refer itching in other places where the skin is under pressure.

Asymptomatic: patient not referred rash after pressure, either referred itching in places that are under pressure effect (bra, underwear, belt).

Erythematous papular phenomenon: it can be explained by the so-called triple response of Lewis. White line occurs in local press, about 30 seconds after the line is erythematopapulous the surrounding areas also become erythematous. White dermatographism: it is an anomalous response that can occur in about 50% of atopic patients. The linear response after pressure exerted on the skin turns white instead of red.

- Non-occupational dermatoses more frequent.

Superficial mycoses.

Mycoses are caused by various fungi that use keratin as a source of Livelihoods of the skin, hair and nails.

Dermatophytes are the major producers of superficial mycoses in humans and comprise three major genres: Microsporum,

Trichophyton and Epidermophyton.

- Key superficial mycoses, tinea cruris tinea pedis and tinea corporis.

The main causative agents of superficial mycoses are: Trichophyton rubrum, Trichophyton mentagrophytes and Candida albicans.

Ringworm of the nails (onychomycosis)

Pityriasis versicolor: Malassezia furfur or Pitirósporum ovale.

Interdigital candidiasis, axillary, crural or periungual that may be aggravated

= In various occupational activities.

- Viral skin.

Herpes simplex, herpes zooster, rubella.

- Urticaria: characterized by the sudden eruption of wheals with short duration.

Major causes of hives.

- Exogenous

Drugs: penicillin and its derivatives, other antibiotics, sulfonamides, analgesics, anti-inflammatory, antispasmodic, psychotropic drugs, laxatives, and other syrups.

Note: acetylsalicylic acid (AA S) is always a strong suspect as the cause of urticaria. Foods: preservatives and dyes, and canned foods in special packaging such as plastic cups, cardboard boxes, plastics and others. Citrus: lemon, sour orange, other acidic fruits such as pineapple,

Grapes, mango, and others. Oilseeds: walnuts, chestnuts, peanuts, coconut, corn, almonds and

Others. Seafood: oysters, shellfish, mussels

-Endogenous: Atopy, intestinal worms, infections, thyroid cancer. Examples of occupational dermatitis in different occupational activities may be showing in the Atlas of Occupational Dermatoses (Salim Amed Ali) released on CD by Fundacentro CTN - Sao Paulo

*** Data: Department of Health Care** Department of Strategic Programmatic Actions

MINISTRY OF HEALTH Occupational Health-Complexity Protocols Differentiated.

CHAPTER 4

LEGAL SUPPORT

Based on the Decree No: 79037, 1976, which regulated Law No: 6367, 1976 (Work Accidents), did include attachments, among them ANNEX I, identifying pathogens and professional activities related to them with special highlighted, recognizing implicitly that the chemicals have on the human body harm.

PATHOGENS:

ARSENIC

Arsenic (from the Greek "arsenikon" is a solid element which is presented in the form metal or nonmetal. In metallic form crystallizes in the hexagonal system and the way in regular metalódica (. There are: a) Symbol "The" b) atomic mass, "74, 91," c) atomic number "33", d) density: 4, 7 "or" 5, 7, e) melting point: 450 "C"

They still have the features like native element presents massive or granular appearance when not in stalactites or reniform structures. Sometimes occurs in veins associadoscom ores of silver, cobalt, nickel, lead or gold, and other minerals such as barite, ciábrio,

PROCEDURE FOR OBTAINING:

a) Arsenic trioxide (white arsenic, arsenic oxide, arsenious oxide, etc.), is obtained as a byproduct of the refining of metals, lying in the fumes and particles from the treatment of minerals cupriferous. From the smoke and dust that condensed material is called "crude arsenic" and contains about 90 to 95% trióxido. Cerca 77% is for the manufacture of pesticides, 18% in the manufacture of glass and 04% in manufacturing chemicals and 1% in the manufacture of medicines.

(B) pentoxide arsenic (arsenic trioxide, arsenic oxide) is employed in the chemical industry for obtaining metal salts (arsenate) and as a defoliant in cotton. can also be utilizado madeira. Arsenito for the conservation of calcium (calcium metarsenito) has been employed como inseticida germicida. Arseniato and potassium (monopotássio arsenate, potassium arsenate diacid) is applied in conservation and leather industries.

POISONING BY ARSENIC

Arsenic is a causative agent of constant occupational poisoning. E used in tanning, Manufacture, formulation and use of ink-based arsenic

compounds, arsenic ores Metallurgy, Processing and preserving of skins and animal plumes. Empalhamento., Determining where frames of chronic evolution.

Absorption can be by inhalation and percutaneous manner.

Signs and symptoms: a) drive: chest pain, dyspnea, bronchitis, chemical pneumonitis, pneumoconiosis, b) gastrointestinal: anorexia, nausea, hypertrophic gastritis, duodenal ulcer, colitis.

Besides irritating can destroy all body cells. Poisoning can be acute or chronic:

In acute are identified: contact dermatitis "nest of a dove," skin ulcer (painless), convulsions, coma, anosmia, nasal foul odor, runny nose, cacosmia, nausea, vomiting, yellowish (bichromate) or hemorrhagic diarrhea, liver necrosis controlubulares, renal damage, oliguria, anuria, decreased prothrombin time, severe uremia.

In the context of chronic poisoning are checked: pyoderma, burns, eczema, nail down, rhinitis, nasal ulcerations asymptomatic predisposition to lung cancer, nasal septum perforation.

DISEASES OF ABSORPTION FROM:

May occur: a) skin b) eyes c) in the lungs, d) in respiratory disorders; e) gastrointestinal;

a) SKIN;

I-Sensitivity to dermatitis (skin diseases and facilitation to get pictures of exacerbation of dermatitis that become more exuberant, vermeridão associated with the skin.

II-skin ulcers, wounds (holes) with lesions of the superficial layer of skin, especially hands, bases of the nails and joints.

b) Eyes:

Conjunctivitis, redness and tearing through irritation of the conjunctiva of the eyes layer.

c) LUNGS:

I-Chest pain.

II-Shortness of breath (dyspnea)

III-Bronchitis (inflammation of the bronchi which causes shortness of breath, wheezing and coughing up secretions).

In inflammation of the bronchi occurs outside liquid blood within the respiratory tree and also decrease the caliber of the bronchi increasing shortness of breath and cough.

IV-chemical pneumonitis, (lung irritation and inflammation resulting from contact with arsenic causing breathing difficulties.)

V-Pneumoconiosis; is a chronic lung disease with fibrosis (thickening caused by scar areas), caused by inhalation of inorganic powders. Usually requires prolonged exposure and there is a great relationship with the immunological part.

Inhalation of certain substances can cause lung inflammation varied by replacing the normal lung tissue by fibrosis (scar areas), destroying other areas (emphysema), leading to respiratory failure and incapacity.

It is irreversible and even removed the aggressive factors pathology persists, but does not regress.

VI-bronchogenic carcinoma, lung cancer is an uncontrolled proliferation of cells

d) BREATHING DISORDERS:

R-injury and ulceration of the nasal septum.

II-perforation of the nasal septum (whistles when the person breathes and the air passing through the small hole. When increasing the drilling area.

III-Epistaxis (nasal bleeding)

IV-Sinusitis: inflammation of the sinuses with extravasation of fluid and white blood cells causing secretion within the frontal sinuses, accompanied by fever, headache and pain in the sinuses.

V-Laryngitis (inflammation of the larynx due to irritation)

VI-Anosmia (absence of smell)

e) Gastrointestinal:

I-Lack of appetite (anorexia)

II-Nausea

III-hypertrophic gastritis (inflammation of the inner wall of the

stomach cells to increase production and secretion)

IV-duodenal ulcer (wound on the inner wall of the duodenum and may cause pain, bleeding and even perforation.

V-colitis

CHAPTER 5

OCCUPATIONAL TUMORS

GENERAL

Tumors professionals may stem from prolonged exposure to carcinogens or result from the action of the cutaneous trauma. In the etiology of tumors, one should not disregard the existence of concomitant causes, such as individual constitution, lifestyle habits, especially regarding chronic poisoning, such as alcoholism, the maintenance of poor nutrition and rest.

You can often detect the early appearance of a tumor, especially that which originates in the skin. The tumor usually arises in the same place would arise if the worker was exposed to cancer-generating agents, although there are exceptions, such as, for example, with tumors of internal organs that appear in certain professions, like that of the dyers.

Carcinogens:

The carcinogens can be divided into physical, chemical and traumatic:

CARCINOGENIC AGENTS PHYSICAL:

The main physical carcinogens are the heat and radiation.

Some tumors professionals are assigned to high temperatures to which workers are exposed.

b) Radiation

Injuries caused by radiation:

UNITS OF MEASURES taking roots

- The display unit is the R (Roentgen, now replaced by COULOMB / KG.

the absorption unit is the rad, known for strength and retrofitted for today GRAY (Gy), representing 100 RADS.

- The REM (Roentgen equivalent man) is a unit of measurement to validate the ability of a radiation damage in tissues. REM was replaced by the sievert (SV) and SV is a = 100REM. In summary:

R-COULOMB Today / KG = Exhibition

RAD - today GRAY (GY) = skin absorption REM - today Sievert (Sv) =

effect or dose equivalent. In dental radiology because of the peculiarities of the irradiated tissue and the quality (KV), the maid taking roots, we can accept that $a = R = 1\text{RAD } 1\text{REM}$.

DOSE taking roots is the total amount of taking roots sent or received by an organism. International stamp entities such as the ICRP (International Commissions on Radiological Protection USA), sought to establish a concept of "DOSE MAXIMUM PERMISSIBLE" usually expressed weekly, DMPS (Dose Tolerance). This dose is currently £ 5 per year, or 0, 1R/semana. There is also what we call ERYTHEMA DOSE, which ranges from 250 R to 750 R, depending on the sensitivity of the individual irradiated. For safety, the exposures used in dentistry should be restricted to half the minimum dose (250 R). Regarding Radiation human, what is important is not whether the dose-skin, but how much reaches the gonads, there is disagreement about the gonadal dose, ranging around 1 / 1000 of the dose-skin.

A major concern about radiation exposure, is the potential risk to the life of the cell. If an ionizing radiation entering a living cell, it can ionize the atoms that compose it. Already queum ionized atom is chemically different from an electrically neutral atom, this can cause problems inside the living cell.

Typically, these problems are not significant. A large percentage of our body is water, and the chance of ionization occurs in water is very large.

When the damage is done to a vital part of a cell, often the cell can repair the problem through internal mechanisms. Each damage to chromosomes and DNA can be repaired. Chromosomes contain DNA, which are important to enable the functions of the body. DNA is a long molecule found in every cell. DNA molecules provide instructions on how each cell should act. If the DNA in a cell is affected, it can not perform their duties properly. The cell may die. Our body can fix problems in the DNA. In fact, every day are about 100,000 fixed damaged chromosomes.

Many problems can arise if corrections are not made quickly. If the damage is severe, the cell may die. It is also possible that the damage adversely affect cell functions and in some cases, the cell creates replicas of itself may generate a mesma.Isto CANCER.

Basically, four situations can occur when radiation enters a cell:

- 1.Radiation can pass through the cell without causing any damage.
- 2.2. Radiation can damage the cell, but she can fix the problem.
- 3.4. Radiation causes so much damage to the cell dies.

The doses of radiation, large doses over a short period are more dangerous than the same doses in a great period. When we are exposed to certain radiation dose in a long time, our body has time to repair the damage. However, if the period is short, the defense mechanisms can not fix the damage and the cell dies.

The damage to the body, can be large if the cell to reproduce. In the case of marrow cells, we have a framework of leukemia.

For women, the problem may be more serious, because if exposure occurs during pregnancy, the risk of mutations in the fetus.

Radiation is a natural phenomenon that can occur in many forms. Depending on the amount of energy, a radiation can be classified as ionizing or nonionizing radiation. Non-ionizing radiation have relatively low energy. In fact, radiações não-Ionizing are always around us. Electromagnetic waves like light, heat and radio waves are common forms of non-ionizing radiation. Since ionizing radiation can alter the physical state of an atom and cause the loss of electrons, making them electrically charged. This process is called ionization. As an example quoted the alpha, beta, neutron, gamma or X-rays.

In the interplanetary radiation environment deserves serious consideration when planning a mission to other planets. On Earth or in Earth orbit, we are protected from radiation (from various sources) by Earth's magnetic field. Since space does not provide the same protection, and, moreover, is very dangerous risk of being exposed to radiation. The radiation in free space can be classified into two types: electromagnetic radiation and ionizing radiation.

Doses of Radiation:

0077 REM: Medical and dental X-rays.

0082 REM / year: Natural background radiation (cosmic rays, radon, etc.).

0, 14 REM / year: dose to normal sea level from cosmic rays and natural radiation from rocks.

0, 5 REM / year: Residents in the vicinity of nuclear power plant.

5 REM / year: nuclear power plant worker.

25 ~ 50 REM: cell death, especially of the lymph tissue.
Exposure to ionizing radiation astronauts limited to a maximum of 25 months by REM and REM for 50 years, not exceeding 400 REM during his lifetime.

50 REM: Typical dose for an expedition of 2, 5 years on Mars, outside the Earth's magnetosphere. But the body can heal itself over time. This level of radiation increases the risk of cancer by 1% per year, about

how smoking for that period.

REM 100: After 03 hours of radiation appears to intoxication, characterized by insomnia, fatigue, general weakness, poor appetite, nausea, mental instability, vomiting, headache, decreased blood pressure, diarrhea, leukemia, moderate, due to decreased the ability of bone marrow to produce blood cells.

75 ~ 200 REM in 30 days. The body is not able to repair the damage so quickly. The radiation sickness (vomiting, fatigue, hair loss, due to defects in children doses during pregnancy, the development of cancer in the future).

400 REM: Median lethal dose that causes death in 50% of the exposed population within 60 days. Two hours after exposure has: atrophy of the spleen, production of blisters and skin ulcers, bleeding, infections, hair loss, leukemia. Therapy: antibiotics and blood transfusions.

500 REM: Fatal dose. 100% of death in 02 days because there is total destruction of the intestinal mucosa. Large solar flares. You can reach more than 2,000 rems per hour.

1 roentgen is approximately equivalent to 50 chest X-ray X. During the life of a human being, supporting a deep tissue exposure from 100 to 400 rem, 400 rem eyes and the skin can withstand up to 600 rem.

Lethal dose for 50% of individuals in 30 days (REM): Ram = 250, 350 = Dog, Man = 450, 600 = Mouse, Rat = 700, 800 = Rabbit, Snail = 20,000, = 80,000 fruit fly, Amoeba = 100,000.

• Infrared rays •

Works with electric welding, welding with oxyacetylene, works with metal and glass bulbs, that is, are the orange and emit light when overheated, and also in kilns, furnaces and drying processes, ink and material activities that are damp produce infrared rays. Work in the open, the worker is exposed to the sun, which is a natural source emitting infrared rays. At doses well controlled, infrared rays are used for medicinal purposes. But when the intensity of this radiation exceeds the tolerance limits, reaching the worker without proper protection, the infrared rays can cause serious damage to health.

• • Ultraviolet rays

Activities with electric welding processes, photo-reproduction, sterilization of air and water, production of fluorescent light works with arch-voltaic devices, used by dentists, aluminothermic processes (chemical activity with the use of aluminum powder), lamps Special emit

ultraviolet rays from the sun. In small doses (about 15 minutes of daily sun exposure), the ultraviolet is necessary because the man is responsible for the production of vitamin D in the human body. But in excessive amounts, can cause serious damage to health.

Both the ultraviolet and infrared rays are not normally measured in the workplace, but when they are activities that emit these rays, such as those cited in this class, protective measures should be taken to ensure the health of workers.

•• Microwave

Microwaves are found in domestic and industrial forms: microwave ovens, radar devices at airports, radiocommunication equipment, equipment for heat diathermy and heating processes in the production of plastics and ceramics. The measurement or evaluation of the microwave can be by thermal or electrical system, but it is not usual and there are no national limits of tolerance set.

• Laser •

The English acronym, stands for "Light Amplification by Stimulated Emission of Radiation" which can be translated into Portuguese for: light amplification by stimulated emission of radiation. The laser beam is a directional convergence, ie, concentrated in one point. It is widely used in steel mills to cut metals, welding equipment and also for measurements over large distances. It also has applications in medicine, to modern surgical procedures.

The dangers that may represent laser beams have been the subject of studies and experiments so far inconclusive. Hence the recommendations are limited to the more preventive aspects. The greatest effect in man is on the eyes, causing major damage to the retina, which is the sensitive membrane of the eye, in some cases irreversible, and can cause blindness. All those studied radiation: infrared, ultraviolet, microwave and laser are classified as non-ionizing radiation.

However, the most dangerous are the ionizing, whose energy is so great that, reaching the human body, produce changes in cells, causing cancer.

•• Ionizing radiation

From the standpoint of the study of environmental conditions, ionizing radiation of concern in industrial use are X-rays, gamma and beta, and industrial use are not the alpha rays and neutrons, each with a range of wavelength 1 . Such radiation can be found naturally in

radioactive elements such as Uranium 238, etc. Potassium 40. In addition to cosmic radiation from space celeste. Artificialmente are brought about by modern technology such as X-rays, used in metallurgy to detect flaws in metal structures and check for faulty welds. Other types of radiation are used to determine thicknesses of sheet metal, glass or plastic, as well as to indicate liquid levels in reservoirs.

Gamma rays are used to analyze welding metal tubes, a process called scintigraphy. Radiation is also used in luminous paints, power plants producing electricity (such as atomic power plant at Angra dos Reis) and the verification processes of wear of floor wax, wear of tools for turning and rings of car engines . They are also used in research laboratories and in medicine in fighting cancer and many other applications. The absorption of radiation in the human body is indirectly assessed by the unit called REM, in English: "Relative Efect Man" which in Portuguese means: effect on humans.

The detection of ionizing radiation is performed by various devices such as personal and scintillation detectors, dosimeters, etc.. The exposure limits are set by the National Commission for Nuclear Energy and the Ministry of Labour rule.

Radiation-light

It has been observed that in certain groups of people who work outdoors, they develop skin tumors, especially on the bare, in percentages higher than normal in other groups.

Farmers, sailors and fishermen due to the labor activity of particular predisposition to this type of tumor. The origin of cancer produced by ultraviolet rays develops from the melanin hyperpigmentation.

CHAPTER 6

THE WORKERS AND ILLNESS

ITS RELATION TO WORK

Recognition of the role of work in the determination and evolution of the health and illness of workers have ethical, legal and technical, which is reflected on the organization and provision of health actions for this segment of the population, the network of health services.

From this perspective, the establishment of causal relationship or nexus between a particular health event - injury or illness - individual and collective potential or installed, and a given working condition is the basic requirement for the implementation of the actions of Occupational Health in health services. Schematically, this process can be initiated by the identification and control of risk factors for health in these environments and working conditions and / or from the diagnosis, treatment and prevention of damage, injuries or illnesses caused by work, the individual and the collective of workers.

While fleeing the objectives of this text, which deals with the pathogenic aspects of work, potentially producing suffering, illness and death, it is important to note that in actuality, they grow in importance for recovery of the positive aspects and health promoters, also present at work that must be addressed in health practices.

In this chapter will be presented in summary form on conceptual aspects of illness among workers and their relationship to work, some of the available tools and resources to investigate the relationship between health and disease and work-to establish the nexus of damage / disease with the work and actions arising that should be implemented. At the end is a suggested bibliography related

Workers share the profiles of illness and death in the general population, depending on your age, gender, social group or insertion in a specific group of risk. In addition, workers may become ill or die from causes related to work, as a consequence of their profession or performed, or by adverse conditions in which their work is or has been made. Thus, the pattern of illness and death of workers resulting from the amalgamation of these factors, which can be summarized in four groups of causes (Mendes & Dias, 1999):

- common diseases apparently unrelated to the work;
- common diseases (chronic degenerative, infectious, neoplastic,

traumatic, etc.). eventually

Modified to increase the frequency of its occurrence or the earliness of its appearance in workers under certain conditions. Arterial hypertension in urban bus drivers in large cities, exemplifies this possibility;

- common diseases that have a spectrum of etiology extended or made more complex by the work.

Bronchial asthma, allergic contact dermatitis, the noise-induced hearing loss (occupational), musculoskeletal diseases and some mental disorders exemplify this possibility, in which, due to work, add to (additive effect) or multiply if (synergistic effect) or the conditions that caused these provocative pictures nosological;

- specific health hazards, typified by work accidents and occupational illnesses. Silicosis and asbestosis exemplify this group of specific insults.

The last three groups constitute the family of diseases related to work. The nature of this relationship is subtly different in each group. Table II summarizes and exemplifies the groups of diseases according to the classification proposed by Schilling (1984).

GROUP I: diseases in which the work is necessary because, typified by diseases, *stricto sensu*, and the acute poisoning of occupational origin.

GROUP II: diseases in which the work can be a risk factor, contributing, but not necessary exemplified by the common diseases, more frequent or earlier in certain occupational groups and for which the causal relationship is eminently epidemiology. Arterial hypertension and malignant neoplasms (cancers) in certain occupational groups or professions, are typical.

GROUP III: diseases in which the work is provocative of a latent disorder, or of aggravating pre-existing or established disease, ie *concausas*, typified by the allergic diseases of the skin and respiratory disorders, in certain occupational groups or professions.

Among the specific diseases included are occupational diseases, for which it is considered that the work or the conditions under which it is performed are the direct cause. The causal relationship or causal connection is direct and immediate. The elimination of the causative agent for control measures or replacement can ensure prevention, ie, elimination or

eradication. This group of disorders, I Schilling, is also a legal concept in the SAT of Social Security and its occurrence must be notified to rules in the sphere of Health, Welfare and Labour.

ERGONOMIC AND PSYCHOSOCIAL:

Stem from the organization and management of work, for example, use of facilities, machinery and furniture inadequate, leading to incorrect postures and positions, adapted to local conditions more lighting, ventilation and comfort for employees, working in shifts and night shifts, monotony or excessive work pace, demands on productivity, labor relations authoritarian gaps in training and supervision of workers, among others;

MECHANICAL AND ACCIDENTS:

Relating to the protection of machinery, layout, order and cleanliness of the workplace, signage, labeling and other products that can lead to accidents.

The resources and technical tools available to investigate the relationship between health-disease work are summarized in Table III. They are organized and presented according to the focus of the investigation, damage and / or risk factors, the individual and the collective of workers. More information about the conceptual and operational aspects, its uses and limitations may be found in the suggested bibliography at the end of the chapter.

Importantly, to investigate the relationship between health-disease-work is essential to consider the report of workers, both individually and collectively. Despite the advances and sophistication of techniques for the study of environments and working conditions, often only the workers know how to describe the real conditions, circumstances and contingencies that occur in everyday life and are able to explain the illness.

CHAPTER 7

BASES TECHNIQUES FOR CONTROL OF RISK FACTORS AND THE IMPROVEMENT OF ENVIRONMENTAL AND WORKING CONDITIONS

The elimination or reduction of exposure to hazardous conditions and improvement of work environments to promote and protect workers' health is a challenge which goes beyond the scope of performance of health services, requiring technical solutions, sometimes complex and costly. In some cases, simple and inexpensive measures can be implemented, with positive impacts and protection to their workers' health and the environment.

The control conditions of health risk and improving work environments involves the following steps:

- identification of conditions present risk to health at work;
- Exposure characterization and quantification of risk conditions;
- discussion and definition of alternative disposal or control of risk conditions;
- implementation and evaluation of measures adopted.

It is very important that workers participate in all phases of this process since, as noted in the previous chapter, in many cases, despite all the technical sophistication, the workers are only able to report subtle differences between prescribed work and work real, explaining the illness and what should be changed to make it produce the desired results.

Nowadays, concern for the environment and the health of residents in the area of influence of the production units has strengthened the movement seeking change of work processes potentially harmful to people's health and the environment, which may be an important ally health worker.

Are presented below with some considerations about the concept of risk and risk factors or conditions for health; the available methodologies for the recognition of risks, some of the alternatives for the elimination or reduction of exposure to hazardous conditions for health and improvement of working environments in order to protect worker health. More information and deepening of these questions can be obtained from the literature related to the end of chapter.

Identification and Risk Assessment of Conditions

The concept of risk used here derives from the English word hazard, which has been translated into Portuguese as a danger or risk

factor or risk. According Trivelato (1998), the concept of risk is two-dimensional, representing the possibility of an adverse effect or damage and the uncertainty of the occurrence, distribution in time or magnitude of adverse outcome. Thus, according to this definition, situation or risk factor is "a condition or set of circumstances that has the potential to cause an adverse effect, which can be: death, injury, illness or damage to health, property or the environment."

Also according Trivelato (1998), the risk factors can be classified according to their nature, in:

ENVIRONMENTAL:

- Physical: some form of energy: radiation, noise, vibration, etc.;
- Chemical, chemicals, dust, etc.;

SITUATION: facilities, tools, equipment, materials, operations, etc.;

HUMAN BEHAVIORAL OR: resulting from human action or inaction.

The recognition of hazardous conditions at work involves a set of procedures designed to determine whether or not there is a problem for the worker and, if so, to establish their likely magnitude, to identify agents of potential risk and opportunities exposure. It is a fundamental step in the process which, although subject to the limitations of available resources and errors, as the basis for the decision on actions to be taken and for establishing priorities. Recognize the risk means, within the working environment, factors or situations with potential for harm, that is, if there is a possibility of damage. Assess risk means estimating the probability and severity of the damage occurs. Once established the causal relationship or nexus between the disease and the work performed by the worker, the professional or team responsible for servicing should ensure:

- guidance to workers and their families about their health problem and the necessary referrals to health recovery and improved quality of life;

- away from work or occupational exposure, if the worker's stay represents a factor of worsening or slow its improvement, or those in which functional limitations hinder the work;

- the establishment of appropriate treatment, including procedures for rehabilitation;

- request to the company issuing the CAT to the INSS, and be responsible for completing the Report of Medical Examination (LEM). This measure applies only to workers employed and insured by the SAT / INSS. In the case of public officials, for example, must be adhered to

specific standards.

- notification to the health authority, through specific instruments, according to the laws of health, state and municipal levels, enabling the procedures of public health surveillance. It should also be reported to the DRT / MTE and the labor union to which the employee belongs. The decision as to absence from work is difficult, requiring numerous variables of medical and social character are considered:

- cases with total disability and / or temporary should be off work until clinical improvement

Or change of function and removal of risk;

- if a worker be kept in operation, must be identified to the alternatives compatible with the limitations of the patient and found no risk of interference in the evolution of its board of health;

- when the damage produced is small, or there are activities consistent with the limitations of the patient and found no risk of worsening of his health framework, it can be reassigned to another activity, or part-time, according to his state of health;

- when there is need to remove the patient from working and / or her usual activity, the doctor must issue a report explaining the reasons for expulsion, forwarding it to the company doctor, or responsible for PCMSO. If there is evidence of exposure of other workers, the fact must be reported to the company and requested corrective actions.

Special attention should be given to the decision to return to work. It is important to assess whether the company or institution offers a program of returning to work, offering activities consistent with the formation and function of the worker, respectful of their possible limitations in relation to stage pre-injury and prepare colleagues and managers to support employee in the new situation by extending the concept of work ability in the company adopted in order to avoid the exclusion of workers in their workplace.

Considering the character of the area of construction worker's health is important for health care professionals are imbued with the responsibility of production and dissemination of accumulated knowledge.

To recognize the conditions of risk is necessary to investigate the possibilities of generating and dispersing agents or harmful factors associated with different work processes, operations, machinery and other equipment as well as the various raw materials, chemicals used to any byproducts and waste. The possible effects of agents potentially present health should be studied.

Thus, the available knowledge about the potential risks that occur

in a particular work situation must be accompanied by a careful observation on the spot the actual conditions of exposure of workers.

It must be remembered that there is a difference between the ability of an agent to cause harm and the possibility that this agent causes harm. The intrinsic potential of a toxic agent to harm health only be achieved if there are conditions for it to reach the agent (s) organ (s) critic (s) he can damage. For example: free crystalline silica is the causative agent of silicosis, so a block of granite "terminating" the risk of silicosis. However, this block offers only real risk of disease if subjected to any process of subdividing the production of particles small enough to be inhaled and deposited in the alveoli. If the block is part of a granite monument, there is no risk of silicosis, but if that same block of granite is in a corner in the workplace is important to investigate to be used. The fact that at present not be offering risk does not mean that they would be in the future.

Some examples, not exhaustive, chemical, physical and biological processes that may pose a risk to health, as well as places where they occur.

The presence of atmospheric contaminants can go unnoticed by setting the hidden risks.

The lack of characteristic properties or the simultaneous presence of a multiplicity of factors in the workplace can mask risks, for example, the odor. When the risk comes from products or substances used is simple.

The problem of impurities must be carefully examined, since some chemicals can contain contaminants more toxic than themselves, posing risks to health. For example, the benzene, a highly toxic and carcinogenic, can be found as an impurity in gasoline and other less toxic solvents such as toluene and the xylene. Some powders contain asbestos as impurity. The arsine and phosphine, very toxic gas, can be found as impurities in acetylene, which is much less toxic.

Products sold under trade names, without detailed information regarding the chemical composition, often create problems for the recognition of risks. Such information should be required of manufacturers and suppliers, since analysis of samples of such products are difficult and expensive. Currently, available data bases with information on products from brand names, including toxicological information. Some of these information sources are referenced in the bibliography at the end of this chapter.

Another important aspect of the toxicity of chemical substances refers to its physical properties. The proportion of the components of a

vapor may differ greatly from their proportion in the mixture which gave it birth.

For example, a mixture containing 10% benzene and 90% xylene in the liquid phase will contain 65% benzene and 35% xylene in vapor phase, therefore, a much larger proportion of the most toxic component. Liquids containing small amounts of impurities are highly toxic, however, with high pressure steam, may give rise to dangerous fumes, if inhaled.

As for dust, its composition may differ greatly in composition of the rock that gave it birth, due to differences in the friability of the components. Also its visual appearance can be deceiving. Visible clouds of dust may be less harmful than almost invisible clouds, because some fraction of respirable dust, the most harmful, can not be seen with the naked eye. Due to its small size and light weight, can stay airborne for a long time and reach great distances, affecting workers who appear not to be exposed.

Another risk, sometimes forgotten, for lack of oxygen, which can quickly lead to death. Can occur when certain atmospheric contaminants does not necessarily toxic in themselves, displace the oxygen, as in an enclosed space where there is fermentation and CO₂ displaces oxygen.

With the exception of ionizing radiation, the risk of physical nature are usually easy to recognize,

Because they act directly on the senses. In Table VIII are listed some examples of physical agents and their exposure situations.

Exposure to biological agents is often associated with work in hospitals, clinical laboratories and agricultural activities, but can also occur in other locations. The fact that frequently occur in non-occupational complicates the establishment of a causal connection. Biological agents include viruses, bacteria, rickettsia *, protozoa and fungi and their spores. In Table IX, presented below, are listed some examples of these agents and their situations occupational exposure.

The factors of illness related to work organization, generally considered in the ergonomic hazards can be identified in various activities, from traditional agriculture to modern business processes that incorporate high technology and sophisticated management strategies. The processes of productive restructuring and globalization of market economy, in progress, have caused significant changes in organization and management of work with major implications for worker health. Among its highlights are the consequences of musculoskeletal problems and mental illness-related work, growing in importance worldwide. The demand for greater productivity, coupled with the continued reduction in

the number of workers, the pressure of time and the increasing complexity of tasks, and unrealizable expectations and labor relations tense and precarious, psychosocial factors are responsible for stress-related situations work.

The recognition of risk conditions present in the work can be accomplished with the aid of various methodologies, but they all include three basic stages:

a) the initial study of the situation;

- Rickettsiae - from the Latin rickettsia (Sing.) / rickettsiae(Pl.):

Any order escotobactéria Ricktsiales

. B)inspection of the workplace for detailed observations;

c) data analysis.

The initial study of the situation is essential if risk factors or conditions are not overlooked during the inspection of the workplace, requiring technical knowledge, experience and access to expert sources and updated information. The preliminary study (s) Case (s) of work, preceding the inspection, it can be done using the available information sources (literature, electronic databases, technical reports of previous surveys conducted in the same place or in similar locations) and through anticipated questions the very company that will be studied, for example, the list of products purchased with their rate consumption (weekly or monthly), how and where it is used. So it is possible to determine the priori what are the main potential risk, which will be of great utility and perfect time during the inspection itself. Completed the investigation of agents of potential risk, which may occur in the workplace, it is necessary to check what are its possible effects on health. It also should be consulted tabulated Occupational Exposure Limits (OEL) or Limits of Tolerance (LT), because the values of permissible exposure to different agents give an idea of the degree of damage they can cause and are useful for make comparisons and set priorities.

For example, a chemical agent whose LT is 0, 5 mg/m³ is much more dangerous than an agent whose LT is 200 mg/m³.

Information regarding the health of the worker, including complaints, observed symptoms or other effects on health and early changes in health parameters or results of biological monitoring can also help identify dangerous conditions existing in the workplace . Close collaboration between those responsible for the study of environment and working conditions (hygienists, safety engineers, ergonomists) and those responsible for worker health (doctors, psychologists, nurses work, toxicologists) is essential for a correct assessment of exposures occupational. The multidisciplinary approach and teamwork help discover

causal relationships that may otherwise go unnoticed.

The potential harm of a particular agent found in the work environment is important for the establishment of priorities, even for the remarks, warning of the presence of serious conditions that require immediate action, such as exposure to very toxic , carcinogenic or teratogenic. The mode of action of an agent on the body (fast, slow) and the ability to penetrate through intact skin are important data to guide the observations on the spot and the establishment of the sampling strategy, if necessary.

Reports and results of previous investigations must be analyzed considering the possibility of changes have occurred in working conditions.

Upon inspection of the workplace is important to set a focal point, which necessarily must be a person who knows the whole process of work, ensuring access to people who can give relevant information, especially the workers. All information gathered should be recorded clearly, in a format prepared in advance, including checklists concerning possible risk factors in each operation. It is essential to obtain or prepare a flowchart of the process.

If it is not possible before, when at the time of inspection of the workplace must be obtained a list of different materials and products purchased and used. Information regarding the consumption rates (weekly, monthly) and how and where they are used can help in establishing the magnitude of the likely risk and the location of resources that might escape notice, particularly if they are hidden. Not always the use of chemicals is apparent. Receiving areas and storage of materials can not be forgotten. Among the questions to be answered are: What substances are used? How many?

How and where? In the case of chemical agents and dust, which the ability of evaporation or dispersal? Aspects to be observed are: production technology and processes, equipment and machinery, potential sources of contaminants, including conditions that could lead to accidental formation, for example, improper storage of reactive substances and circumstances that can influence their dispersal in the workplace, and the likely direction of spread of these contaminants from the source.

Possibility of leaks and fugitive emissions in closed or isolated cases should be carefully investigated. Among the questions to be answered are: what are the sources of emissions?

It is necessary to process? The task can be performed with less risk? What is the worker? In the case of closed proceedings, the

possibility of fugitive emissions?

It is important to ask about sporadic cases that may not be running at the time of inspection. All cycles of the process should be investigated and, preferably, observed. Workers can give valuable information in this regard.

The general characteristics of the workplace and the possible influence of contiguous environments must also be observed. Example: poisoning can occur by exhaust gases from vehicles left with the engine running on a platform for loading / unloading adjacent to open windows in a workplace where there are no harmful air contaminants. Even more serious situations can occur and have occurred when toxic contaminants are carried by wind or by an escape, entry points for air ventilation systems. The layout the environment should be noted, the posts work and the tasks must be observed and analyzed.

In addition to studying the possible occurrence of dangerous conditions in the workplace and the harm they can cause, it is necessary to observe the conditions of exposure, which include features such as routes of entry into the body, physical activity level and exposure time. The investigation of exposure conditions is also necessary to define the sampling strategy, for a correct quantitative evaluation and planning of prevention and control. About the ways into the body of chemicals and dust is important to consider that, in the workplace, the airway is the most important. It is influenced by the manner of the worker's breathing if the nose or mouth and by type of activity, since the heavier work requires more ventilation.

At rest, a person breathes, averaging 5 to 6 liters per minute and to perform very heavy work will breathe 30 to 50 liters per minute. In the case of dust, the mechanism of existing filters in the nose is important and can occur an appreciable difference between the amount of dust inhaled and deposited in different regions of the respiratory tract, depending on the type of breathing if nasal or oral. Mouth breathing increases the deposition of respirable dust in the alveolar region, compared to breathing through your nose. The degree of physical activity also has great influence, significantly increasing the deposit of dust in all regions of the respiratory system.

Some substances can be absorbed through intact skin and through the bloodstream, contributing significantly to the total absorption of a toxic agent. Characteristics of the chemicals that influence the absorption through the skin include solubility (more soluble in lipids, higher absorption) and molecular weight (the higher the lower the absorption). Other factors influencing absorption include the type of skin, which varies

from person to person and also a body part to another, the skin condition, such as the existence of skin diseases, like eczema and fissures; prior exposure to solvents and heavy physical work, which stimulates peripheral blood circulation. It is important to investigate, among the potential agents of exposure, which have the property to be absorbed through the skin. Some chemicals in the form of granules or flakes can offer such a risk, if any direct contact with skin and if they are soluble in sweat, for example, pentachlorophenol. This situation is aggravated in hot workplaces. The possibility of absorption through the skin modifies the procedures for the assessment of exposure by simple quantitative sampling / analysis of air, not will be sufficient to evaluate the overall exposure.

Also, control by means of respiratory protection will not be sufficient to protect the worker, who shall incorporate appropriate work practices and avoid skin contact and splashes on clothing and institute strict personal hygiene.

Although the gastrointestinal tract is the least important entry in occupational situations that possibility should be investigated and eliminated through the establishment of work practices and hygiene.

The level of physical activity required is paramount, too, in case of thermal overload, because the more intense the higher the metabolic heat production that must be dissipated.

The evaluation of the dose actually received by the worker is an agent of a chemical or physical agents present in the work situation, depends on the concentration, when it comes to an atmospheric contaminant, or intensity, when referring to a physical agent, and exposure time. Examples: compliance with standards, noise exposure should not exceed 85 dBA for an occupational exposure of eight hours daily, but can go to 88 dBA for 4 hours per day or 91 dBA for 2 hours daily. Heat exposure in an environment with Wet Bulb Index - Globe Thermometer (WBGT) equal to 29.5 °C for moderate work is not acceptable for continuous work, but it would be for a scheme of 50% work and 50 % of rest in a cool, per hour or 30 minutes of work, 30 minutes of rest.

For chemical agents, the influence of exposure time varies for agents of fast action in the body or those of chronic action. When the action is fast, even short exposures should be avoided. Exposure to carcinogens and teratogens should be eliminated and be under strict control.

On the fluctuations in the conditions of exposure to chemicals, in most cases, the release of air pollutants varies with place and time. Appreciable fluctuations and possibilities of occurrence of peak levels of

air contaminants must be observed in the processes and variables in sporadic operations such as opening oven drying or polymerization reactors. This information is crucial for the development of sampling strategies in quantitative evaluation and planning of measures for prevention and control that in some cases, should aim at a specific phase of the work process, for example, respiratory protection at the opening of a drying oven.

The number of exposed workers to be protected influencing the choice of methods and economic considerations. While few workers are exposed, may be acceptable to control the exposure through the use of Personal Protective Equipment (PPE) with limited exposure and under strict medical supervision. But we can not forget that the environment is a whole and even if few workers are exposed to noxious agents may leave the workplace to the outside and cause damage to the surrounding communities and the environment in general, demanding to be controlled at source .

The existing control systems, eg, local exhaust ventilation equipment and other systems may exist, should be carefully examined to avoid false security. Closed processes should be tested for leaks and fugitive emissions. The existence of an exhaust ventilation system does not mean that there is effective control, because the system may not be functioning properly.

Applications must be made accountable to the plans and schemes of verification and periodic maintenance of the system, because if this is not done routinely and correctly, even excellent systems initially, over time, lose their effectiveness. It should also be observed if the contaminants are not being thrown from the desktop to the outside environment. The availability of PPE for workers does not mean they are protected, because the equipment may not be effective. In the case of masks for respiratory protection, for example, they can not be adjusted, may have leaks, filters can be worn or inadequate. Particle filters do not serve in the presence of vapors. No filter is used if there is lack of oxygen.

In certain situations tools can be used for the recognition of hazardous conditions, for direct reading, useful for an initial screening and verification of the presence of a particular agent in the atmosphere.

Although the results are not very accurate and precise, may serve to elucidate suspected hidden risks. And semi qualitative assessments may be sufficient at this preliminary stage. Special care should be taken as to whether false negatives, particularly when dealing with potential exposure to agents very dangerous, highly toxic, carcinogenic or teratogenic effects, for which even very low concentrations are

significant. In such cases, the lower limit of detection is critical.

Very sensitive instruments can not register concentrations very low, leading to an erroneous assumption of zero exposure instead of zero detection, which can have serious consequences for trabalhadores. Além addition, one must be careful with other interference that can mask the results.

One should not neglect the protection of the people who make withdrawals, they may be exposed to serious risks, for example, lack of oxygen, high concentrations of H₂S when entering a confined space or cancerous. Must have at its disposal adequate PPE and direct reading instruments to test, before entering hazardous atmospheres. These procedures can be educational for businesses and workers.

Completed the inspection of the workplace, it is essential to draft the report. This should be objective and accurate, clearly indicating the characteristics of the workplace, the name and coordinates of the focal point in the company, all hazardous conditions observed and other relevant factors. Must be drafted so that others may have a clear idea of the situation.

The analysis of information should guide the establishment of priorities and definition of further actions that are, in principle, the following:

- risk if the condition is evident and its potential to cause harm to the environment is severe, this

Recognition should be sufficient to recommend that immediate preventive action, without waiting for the process of quantitative assessment of exposure, usually long and expensive. This is admittedly dangerous for operations, for example, the use of sandblasting, post transfer very toxic, electric welding in confined spaces, spray pesticides, molten metal transfer, which are carried out without the necessary control;

- if it is shown that there is no risk, no need for quantitative assessment of exposure, however, may be noted that any future changes could alter risk;

- if the risk is unclear, a quantitative assessment is needed to confirm and determine the magnitude of risk conditions. Qualitative assessments for decision making regarding the prevention and control have received attention

Increasing due to the fact that it is impossible to make quantitative assessments correct in all situations, and

They are much more expensive and lengthy. However, qualitative assessments should follow an appropriate methodology, for example,

Banding Approach, developed in England, which is a guide for decisions on control measures for airborne contaminants, without using quantitative assessments and comparisons with Occupational Exposure Limits (HSE, 1999). The idea is to estimate the degree of risk from toxicological, quantities of substances used, the possibility of leakage or evaporation and conditions of use and exposure. The information obtained is compared with previously developed tables that indicate the necessary controls. In severe and complex, are recommended to consult experts in prevention and control of risks.

The proposed approach to ergonomics work analysis differs from the methodology used by Occupational Health. The foundations of their practice based on the study of work, particularly the identification of differences between prescribed work and the actual work, which often explain the illness among workers.

The growing complexity of new work processes, organized from the incorporation of technological innovations and new management methods, has generated different forms of suffering and illness of workers, particularly in the mental sphere. In many such situations, the requirements of the Occupational Hygiene classic has been met, but remain present or are added other conditions of ergonomic and psychosocial risk arising from work organization, responsible for the production of illness.

CHAPTER 8

IDENTIFICATION AND CONTROL OF RISK FACTORS IN THE PERSPECTIVE OF THE HYGIENE OF WORK AND ERGONOMICS

The basic principles of control technology proposed by the Occupational Hygiene can be stated as:

- a) prevent a potentially dangerous or toxic agent for health is used, formed or released;
- b) if it is not possible, contain it so it does not spread into the environment;
- c) if it is not possible or sufficient, to isolate it or dilute it in the workplace and, ultimately,
- d) block the routes of entry into the body: respiratory, skin, mouth and ears, to prevent a

Noxious agent reaches a critical organ, causing injury.

The chain of transmission of risk must be broken as early as possible. Thus, the hierarchy of controls should look for, sequentially, the control of risk at source, in the control path (between source and receiver) and, if the former fails, control of risk exposure in workers. When it is not possible, which often occurs in practice, the goal becomes the maximum reduction of the offending agent, so as to minimize the risk and its effects on health.

The information and worker training are important components of preventive measures related to work environments, particularly if the mode of doing things leads to the formation or spread of pests to health or affect the conditions of exposure, for example, position in relation to the task / machine, the possibility of absorption through the skin or ingested, the greater expenditure of energy, among others.

In special situations, can take measures to limit worker exposure by reducing exposure time, specific training and use of PPE.

The strategies to control risk should aim primarily to prevent, through measures of process engineering to introduce permanent changes in the environments and working conditions, including machinery and automated equipment to dispense the presence of the employee or any other person potentially exposed.

Thus, the effectiveness of the measures will not depend on the degree of cooperation between people, such as the use of PPE.

The main objective of control technology should be the

modification of risk situations, through projects adequate and engineering techniques that:

- eliminate or reduce the use or training of agents harmful to health, for example, substitution of materials or equipment and modification of processes and ways of managing the work;

- prevent the release of such agents in the workplace, for example, closed systems, enclosure, local exhaust ventilation, general ventilation dilutive, proper storage of chemicals, among others;

- reduce the concentration of these agents in ambient air, for example, diluting the local ventilation and cleanliness of workplaces.

All possibilities for control of risk conditions present in work environments through Collective Protection Equipment (CPE) must be exhausted before recommending the use of PPE, particularly with regard to respiratory protection and hearing. The control strategies must include procedures for environmental monitoring and occupational health. The health surveillance must contribute to the identification of hypersensitive and workers to detect flaws in the systems of prevention. The information and worker training are essential components of preventive measures relating to work environments, particularly if the mode and perform the tasks favor the formation or spread of pests to health or affect the conditions of exposure.

Summarizing, the steps to define a control strategy include:

RECOGNITION AND EVALUATION OF AGENTS AND FACTORS THAT MAY OFFER HEALTH RISK AND THE ENVIRONMENT, INCLUDING THE DEFINITION OF IMPACT:

Should be determined and localized sources of risk, the possible trajectories of propagation of the agents in the workplace, or the action points of entry into the body, the number of exposed workers and the existence of health problems among workers exposed to the agent. The interpretation of the results will possible to know the real risk to health and setting priorities for action;

DECISION MAKING: Results from the recognition that there is need for prevention, based on information obtained in the previous step. The selection of control options should be fair and realistic, taking into account the technical and economic feasibility of its implementation, operation and maintenance, as well as availability of human and financial resources and existing infrastructure;

PLANNING: once identified the problem, the decision to control it, determine the priorities for action and available resources must be drawn up a detailed design of the measures and procedures

Prevention to be adopted;

EVALUATION.

On the organizational and management to be adopted aimed at improving working conditions and quality of life of workers, particularly for the prevention of mental disorders and mental distress and work-related RSI, it is suggested that they be consulted in capítulos of (Mental and Behavioral Disorders Work Related) and (Diseases of Musculoskeletal System and Connective Tissue Related to Labor). With regard to working conditions hazardous to health arising from the organization and management of work, the recommended measures can be summarized as follows:

- increase in actual control of tasks and work by those who perform them;
- increasing the real participation of workers in decision making processes in the company and its facilities for organization;
- enrichment of tasks, eliminating repetitive and monotonous activities and overtime;
- stimulus situations that allow the worker a sense of belonging and / or forming part of a group;
- developing a relationship of trust between workers and other group members, including superiors;
- stimulus conditions that give rise to the replacement of competition by cooperation

What is the difference between being covered or not covered by the SAT?

Pension legislation in force (Federal Law and Decree No. 8.213/1991 3.048/1999) provides that all insured for Social Welfare (General Scheme), in the case of common diseases, they are entitled, amongst others,

Following benefits and services:

- sickness benefit;
- aid, accident;
- disability retirement.

Both the sickness benefit (benefit in kind, payable from 16. Day of temporary labor incapacity recognized by medical INSS) as a disability

retirement in the case of common diseases, require the grace period of 12 monthly contributions.

Diseases related to work, when put in the requirements of Articles 19 and 20 of Federal Law No. 8.213/1991, are treated as accidents. Currently these are listed in Annex II of Decree No. 3.048/1999 (Lists A and B), as mentioned in the introduction and first chapter of this manual.

Being featured the work accident, for purposes of Social Security, the insured and their dependents are entitled to benefits (sick pay, said the accident, disability retirement allowance, accident, death pensions) and services (social service and vocational rehabilitation) peculiar to this type of health problem, no grace period, ie independent of time of contribution.

With regard to the sickness benefit (monthly income while persisting inability to work, assessed by medical INSS), the only difference between the common disease for granted and awarded by an accident at work (including, therefore, occupational diseases and other work-related diseases) is the necessity of grace. The amount of sickness benefit is 91% of salary benefit, and this "Is the simple arithmetic average of all past decontribuição wages for the months immediately preceding the removal of the activity or the date the application ..." (Article 32 of Decree No. 3.048/1999).

An important outcome of the characterization of a disease related to work by SAT / INSS is stability in employment, one year, that workers get insured after termination of welfare benefits, the accident, that guarantee is not provided after the cessation of aid- common illness or social security (article 346 of Decree No. 3.048/1999).

What are the implications arising from the social security medical diagnosis of a disease related to work?

The diagnosis of work related disease in workers insured by the SAT of Social Security requires that if this has not been done, to open a CAT, Social Security document. The CAT, as a communication tool in the context of Social Security must be completed in the first part, by the company.

Under art. Decree No. 336 of 3.048/1999, "In the absence of communication by the company, may formalize it himself injured, their dependents, the employees union, the doctor who attended or any public authority, not prevailing in such cases, the period mentioned in this article. "(Paragraph 3. of the same article, emphasis added). The deadline for reporting is up to the first working day following the occurrence and,

in case of death, immediately to the competent authority, under penalty of fine. The second part of the CAT, Report of Medical Examination must be completed in the corresponding fields, the doctor who attended the worker, ie, the physician doing the diagnostic work-related accident strictly speaking, of course accident or occupational disease or work, recording his opinion, even if preliminary, as to whether or not off work. It is being implemented by the Social An alternative is to make CAT electronically

CHAPTER 9

5.1 DIAGNOSIS OF A DISEASE RELATED TO WORK AND REMOVAL OF WORK

Many diseases, related or not to work, require for its severity, the immediate dismissal from work, as part of treatment (rest required) and / or the need to stop the exposure to risk factors present conditions and / or environments work. Other diseases, which are less serious, not necessarily imply the absence from work. Many physicians find themselves cast doubt on the issue of medical certificates. Some are very liberal and, certainly, granting extended time off, trying to protect the worker. Others are too stringent or restrictive, giving insufficient time for effective improvement of patient / employee. There is no fixed formula for such a decision, which is at the discretion of the physician attending the patient / employee. The main difficulty stems from the lack of objective criteria to guide the conduct of the physician, especially when he is not familiar with the environment and working conditions of the patient. Thus, some guidelines and information are important:

- not being insured workers, the medical certificate is only a personal document of the patient / employee has not, in principle, another meaning in the case of self-employed, businessmen and sundry;

- for public employees hired under the RJU, the medical certificate of incapacity for work is necessary for him to obtain the allowance of absence from work;

- the worker is insured by Social Security, the medical certificate of incapacity for work will serve to justify his absence from work, by the time the medical request. But in fact, the medical certificate will justify absence from work in just the first 15 (fifteen) days, which are always paid by the company;

- is important to distinguish the move away from the function or activity away from work. The latter situation is almost always linked to the nature and severity of illness and, especially, need for rest, sometimes in bed.

With the necessity of removal of more than 15 (fifteen) days, patient / worker / insured must be submitted to the Forensic Medicine Division of Social Security, where the medical expert will decide on the need for removal,

Arising from the existence (or not) of an incapacity to work. If this

is detected or recognized, trigger the grant of the sickness benefit (Forensic Medical Examination-Initial or Ax-1), whose value corresponds to 91% dosalário benefit. Therefore, from the 16. Day, confirming the need for absence from work, payment

Be borne by the INSS, as long as the disability (temporary) labor. The granting of aid, accident leave for accidents at work, which includes related diseases

Work in Schedules A and B of Decree No. 3.048/1999, due to temporary work incapacity of more than 15 (fifteen) days, ensures the patient / worker / insured stability in employment one year after its termination. It is important to distinguish between disability, impairment and disability for work. The work-related illness or accident at work, in its broadest sense, may have produced or are producing deficiency or dysfunction (impairment), which, according to WHO, is any loss or abnormality of structure or function of psychological, physiological or anatomical . For example, after a Cerebrovascular Accident (CVA), the paralysis of the right arm or dysphasia are deficiencies or malfunction, ie systems or body parts that do not work and that will eventually interfere with activities of daily living normal, producing, in this case, disability. Assessing the nature and degree of disability-if present - is a medical procedure. This assessment can and should be made by the doctor who treated the patient / insured and in a complementary manner, by other experts. The good practice of this procedure aims to avoid the characterization of bipolar all or nothing, using the maximum scales of stages of disability or dysfunction. Several specialties have developed their own criteria. Other references using foreign or international, for example, Guides to the Evaluation of Permanent Impairment, developed by the American Medical Association / AMA (4. Nd edition, 1995) or Bahrain International Disability (y Valoración de las disabilities Del bodily harm, Masson, 1997). As a principle, we seek to, increasingly, to maximize the residual capacity of the patient / employee in all spheres of life including at work.

Already incapacity (disability), according to WHO, is "any reduction or lack (resulting from a deficiency or dysfunction) of the capacity to perform an activity in a manner that is considered normal for humans or that is within the range considered normal. " Refers to things that people can not do. For example, after a stroke that produced the aforementioned deficiencies or malfunctions, the person may be unable to walk, dress or drive a car. For pension purposes, is valued labor incapacity or inability to work, which was set by Social Security as "the impossibility of performing the functions of a specific activity (or

occupation), as a result of morpho psychophysiological caused by illness or accident. (...)

For the vast majority of situations, Social Security works only with the definition, understanding disability as inability to achieve the average yield achieved under normal conditions by workers of the category of the person examined. In the assessment of incapacity to work, you must always bear in mind that the reference point and basis of comparison conditions that must be examined as he worked himself and never

"The collectivity of the average worker." The medical expert from the INSS, in his statement about the existence (or not) of labor incapacity of the insured, consider the following information:

- disease diagnosis;
- nature and degree of disability produced by disease or dysfunction;
- kind of activity or profession and its requirements;
- indication or need for protection of the insured patient, for example, against reexposições
- Occupational sensitization to pathogens or cumulative effect;
- hipersusceptibilidade possible existence of the insured to the pathogen related to the etiology
- Disease;
- relevant legal provisions (such as the Regulatory Norms of the Ministry of Labor and Employment
- (Or, organ health, collective bargaining agreements, among others);
- age and education of the insured;
- susceptibility of the insured or prospective vocational rehabilitation;
- labor market and other exogenous factors.

On technical grounds, to an incapacity to work can be classified into:

- total or partial;
- temporary or indefinite;
- uniprofessional;
- multiprofessional;
- oniprofessional.

However, current pension legislation does not address all of these alternatives and it is expected that the medical expert to rule on Social Security:

- the existence (or not) of an incapacity to work in the short term and on the granting of the relevant social security benefit, sickness

benefit, as regulated by arts. 71 to 80 of Decree No. 3.048/1999;

- the granting (or not) of aid, accident, "Awarded as damages, the insured employee, except domestic, independent worker, insured and especially when the resident doctor, after stabilization of injuries caused by accidents of any kind resulting permanent side effects" that fits the conditions laid down by art. Decree No. 104 of 3.048/1999;

- the granting (or not) of disability retirement due to an insured that, "whether or not the enjoyment of the sickness, is deemed unable to work and subject to no rehabilitation for the financial activity that ensures the survival, conditions established by arts. 43 to 50 of Decree No. 3.048/1999. O medical expert INSS should also proceed to the "technical recognition of the causal connection between the accident and injury, sickness and work accident and the cause and motive" (art. 337 Decree No. 3.048/1999). In other words, will confirm whether a causal relationship or causation, suspected or determined by the doctor who treated the patient / insured in the health service there in the real conditions of work, past or fitting into the SAT. Do not just say "lead poisoning". It will determine whether the poisoning and occupational exposure occurs or if this exposure occurred at work, employment or activity performed by the insured. This procedure may take a trip of the medical expert to work (Resolution / INSS No. 149/1993). It can be seen therefore that, with regard to work-related diseases are distinct roles and responsibilities of the doctor, depending on their institutional context. In general:

- the physician assistant or inserted medical care, the employee will make the diagnosis of disease involving the suspect or establish a causal disease or work-causation, define the treatment, when appropriate, and initiate procedures necessary for the insured patient may enjoy pension rights (in the form of benefits and services);

- the medical expert of the Social Security will evaluate the existence (or not) of incapacity for work, its extent and duration, confirming (or not) by the technical recognition, the causal link between disease and work;

- will fit other roles for doctors working in the company (which would also suspect, or diagnosis of disease related to work) for medical surveillance of the MTE and the doctors who work with the strategy of public health surveillance. All these powers and functions complementamse each other and are equally important.

Instead of celetistas for which CAT is a tool for reporting work-related injuries in case of civil servants governed by their own schemes of work, there is usually instrument specific notification. The federal public servants employed by RJU, victim of an accident at work, must have an open process in the plant or organ in which it works and should be examined by medical expertise, who should characterize the nexus and the potential inability to work. The RJU does not provide specific benefit to the individual victimized by these diseases, except retirement with full salary in the period of total disability.

CHAPTER 10

WORK-RELATED DISEASES

INFECTIOUS DISEASES RELATED WORK

(Group I of ICD-10)

6.1 INTRODUCTION

Infectious and parasitic diseases related to work have some characteristics that distinguish them from other groups:

- etiological agents are unlikely occupational;
- the occurrence of the disease depends on the conditions or circumstances on which work is performed and the occupational exposure, which promotes contact, contagion or transmission.

The etiologic agents are usually mentioned in the name of the disease and are common to infectious and parasitic diseases not related to work. The etiologic agents are widespread in the environment, dependent on environmental conditions and sanitation and the prevalence of infectious diseases in the general population, vulnerable to the general policies and monitoring the quality of health services. The demarcation between the work environment and external environment is often unclear.

The health consequences of worker exposure to biological risk factors present in work situations include pictures of acute and chronic infection, parasitic infections and allergic reactions and toxic to plants and animals. Infections can be caused by bacteria, viruses, rickettsiae, chlamydiae, and fungi. Parasitic infections are associated with

Protozoa, helminths and arthropods. Some of these infectious and parasitic diseases are transmitted by arthropods that act as intermediary hosts. Several plants and animals produce allergenic substances, irritating and toxic with whom employees come into contact, directly, by dust containing animal hair, pollen, spores, fungi or insect bites and stings. In health care workers is important to expose direct patient and the secretions and fluids. Many of these diseases are zoonoses originally, which may be related to work. Among the groups most at risk are workers in agriculture, health (in contact with patients or contaminated materials) in health centers, hospitals, laboratories, mortuaries, engaged in field investigations and surveillance health, vector control and those dealing with animals. May also be affected people working in habitat wildlife, such as forestry, fishing activities, production and handling of animal products, such as abattoirs,

Tanneries, slaughterhouses, food industry (meat and fish) and

workers in sanitation and garbage collection.

Given the extent of exposure situations and the endemic nature of many of these diseases, it is sometimes difficult to establish the relationship to work.

The prevention of infectious and parasitic diseases related to work based on the procedures for health surveillance of workers: epidemiological surveillance of diseases and health monitoring of working conditions and environments, using medical knowledge, clinical epidemiology, occupational hygiene, ergonomics,

Toxicology, among other disciplines, the workers' perception about their work and health standards and regulations. These procedures include:

- measures of education and information to workers about the risks and health effects, modes of transmission and control of agents involved;
- sanitary conditions and working environments through the study of the activities of potential risk to biological agents;
- epidemiological surveillance of diseases, with confirmation of clinical diagnosis of disease and

Establishment of a causal work;

- identification of general and specific measures necessary to eliminate or control exposure to risk factors and for protection of workers;
- control the occurrence of these illnesses in the general population, since a high prevalence of the disorder contributes to increased risks for workers.

Preventative measures vary with the specific disease. The increased incidence of some diseases such as tuberculosis, Hepatitis B and HIV infection healthcare workers has increased awareness of the need for intervention.

From the diagnosis of disease and the establishment of the connection between work should apply to the following:

- assessment of the need for removal, temporary or permanent worker exposure, sector of employment or work as a whole;
- monitoring progress, recording the clinical worsening, relating to, if any, with the return to work;
- notification of this disease to the information system of current morbidity, SINAN or other similar, adding that some of these diseases are notifiable. Can also be reported to the Regional Labor Office and the union of the category to which the employee belongs;

- If the worker is insured by the SAT of Social Security, should be requested to issue CAT, filled the LEM for referral to the INSS;

- active search for other cases in the same establishment or work in other companies of same industry in the geographic area;

- Inspection of the workplace and in other companies of same industry in the geographic area, identifying the factors related to exposure to specific agent and other agents, and the conditions of the working environment, forms of work organization and conduct of activity, technology, tools and machinery used, including equipment, measures and collective and individual protection adopted;

- identification and recommendation of protective measures to be adopted, to inform them

Workers and the employer.

For prevention of exposure to blood-borne pathogens, the Standards for Biosafety precautions or Universaisgprescribe:

- avoid direct contact with body fluids: blood, cerebrospinal fluid, semen, vaginal secretions, breast milk. The others, such as saliva, tears, sweat, urine and amniotic fluid are not considered broadcast media;

- use gloves in the presence of any of these fluids. The use of gloves is mandatory to perform venipuncture, because of the risk of leakage of blood is very great, and to perform invasive procedures such as intravenous injection, intramuscular, collect blood, pass catheter, nasogastric and tracheostomy;

- in case of contact with these fluids by mouth, rinse and mouthwash with hydrogen peroxide 3%;

- if contact with skin, remove the fluids carefully washing the area with soap and water germ. Avoid using brushes, because it causes skin scarification, expanding the port of entry. The skin should be intact with no abrasions or cuts. If the professional you have any skin lesion, it should be covered with waterproof dressings;

- wear masks during procedures where there is the possibility that blood and other bodily fluids reach mucous of the mouth and nose and wear glasses for eye protection, especially in surgical procedures, endoscopy and dialysis;

- Use protective aprons during procedures where there is the possibility of contamination of the clothes of workers with blood or body fluids;

- avoid needle prick injury and causing interruption. Not recapping needles, because this is a risky procedure. Collect the needles in an appropriate solution with sodium hypochlorite at 0, 5% and then put them in the trash. If there is needle prick, immediately press to expel the blood,

wash with soap and water to make soap and occlusive dressing;

- Always wash hands with soap and water and dry them after each patient care, including to administer care in bed;

- taking care of trash and destiny. The hospital waste must be collected in plastic bags, tied and

Packaged in a new bag more sturdy, bound and sent for incineration. The

Responsible for garbage collection shall be vested with gloves, apron and boots;

- take care of cleaning products, utensils and linens. If there is a body fluid spill on the floor, countertop or table, playing sodium hypochlorite 1% in place for 30 minutes;

- manipulate the clothes carefully, without stirring. Collect them and label contaminated. For washing clothes contaminated fluids, use detergent and water at 71 ° C for 25 minutes. In the case of lower temperature than water, soaking in sodium hypochlorite at 0, 5% for 30 minutes.

6.3 LIST OF INFECTIOUS DISEASES RELATED TO THE WORK IN ACCORDANCE WITH THE ORDINANCE / MS No. 1.339/1999

- Tuberculosis (A15-A19 and. -)
- Anthrax (Anthrax) (A22. -)
- Brucellosis (A23. -)
- Leptospirosis (A27. -)
- Tetanus (A35. -)
- Psittacosis, ornithosis, a disease of poultry keepers (A70. -)
- Dengue fever (classical dengue) (A90. -)
- Yellow fever (A95. -)
- Viral hepatitis (B15-and B19. -)
- Disease by the human immunodeficiency virus (HIV) (B20-and B24. -)
- Ringworm (B35. -) and other superficial mycoses (B36. -)
- Candidiasis (B37. -)
- Paracoccidioidomycosis (South American blastomycosis, Brazilian blastomycosis, Lutz's disease) (B41. -)
- Malaria (B50-B54 and. -)
- Cutaneous leishmaniasis (B55. 1) or mucocutaneous leishmaniasis (B55. 2)

6.3.1 TUBERCULOSIS ICD-10 and A15-A19. -

1 DEFINITION OF DISEASE - DESCRIPTION

Disease and its acute, subacute or chronic undertakes various organs and systems, especially the lower airways. In Brazil, the apparent infection *Mycobacterium tuberculosis*, usually transmitted by inhalation, and the *Mycobacterium bovis*, transmitted by ingestion of infective material. It is classified as pulmonary and extrapulmonary forms serous, miliary, uvea, meningoencephalitis, lymph nodes, liver, intestine, kidney, adrenal, osteoarticular and spinal column.

The incubation period is 4 to 12 weeks after infection, with development of positive tuberculin reaction. Most new cases of the disease occurs in 6 to 12 weeks after the incubation period. It is transmitted while the patient is eliminating bacilli. After initiation of therapy in two weeks, the bacilli tend to be more infective.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Worldwide, it is estimated that about one billion people have active tuberculosis, with 8 million new cases per year and 3 million deaths annually. The tuberculosis occurs:

- primary infection due to excessive number of bacilli and / or decreased ability of the host immune response. There are unfavorable to the host immune imbalance;

- in recrudescence, which occurs in about 10-15% of infected people, half of them in the first 2 years after initial infection. In general, due to the reduced capacity of host resistance, and secondarily to a new load of infection by endogenous reactivation. By means of high prevalence of the agent loads new exogenous infective may play a role in secondary tuberculosis. The infection can also be determined by mutant strains of bacilli more virulent multiplicative capacity, or multidrug resistant, with or without immune deficiency (associated with the use of corticosteroids, antitubercular, radiotherapy, immunodeficiency diseases such as in AIDS).

In some workers, tuberculosis can be considered a work related disease, the Group II Classification Schilling, given that working conditions may favor the exposure *M. Tuberculosis* or *M. bovis*, as in the case of workers in biology labs and activities that provide direct contact with contaminated products or with infectious TB. In workers exposed to dustsilica and / or patients with silicosis, tuberculosis and silico-tuberculosis should be considered work-related diseases, the Group III of the Classification of Schilling, once

What has been demonstrated clinically and epidemiologically, that

exposure to silica can promote the reactivation of latent tuberculosis infection, because the crystals silica within alveolar macrophages depress their phagocytic function and increase their destruction.

3. CLINICAL AND DIAGNOSTIC

There is great clinical variability, from asymptomatic cases, insidious and subtle findings, the patient does not know precisely determine the onset of symptoms until significant repercussions acute, subacute or chronic. The clinical picture is characterized by cough, sputum initially less productive and progressively more intense yellow, with hemoptysis or hemoptysis (rare), dyspnea, chest pain, weight loss, fever and sweating (usually evening or night). The pleuritic pain may result from infection of the pleura often associated with the presence of serous cavity effusion in this.

Diagnostic criteria for pulmonary tuberculosis:

Suggestive;

CHEST X-RAYS: shows involvement predominant upper and posterior segments of the lungs, characterized by reticulonodular infiltrates, mediastinal adenopathy, thick-walled cavitary lesions, acinar, miliary, pleural, active and sequels, including retraction of the parenchyma, with loss of lung structure, incarceration;

ANALYSIS OF COMPUTED TOMOGRAPHY:

Expands the vision and radiographic lesions may show no noticeable X-rays, including the presence of lesions suspected of malignancy located in areas of conjunctive neoformation and fibrosis;

Tuberculin skin test (PPD):

Results greater than or equal to 10 mm (strong reactor). The tuberculin test is indicated as an auxiliary method in diagnosing tuberculosis in non-vaccinated with BCG. The positive test, alone, only indicates infection, not necessarily the disease tuberculosis;

Bacteriology and

STUDY Pathology:

Of pulmonary secretions (sputum, for at least 3 samples), other body fluids (tracheobronchial aspirates, washed and gastric lavage), biopsy material, on direct examination, culture or injected into experimental animals, allow a definitive diagnosis of the meeting agent or through PCR amplification or nucleic acid chain.

The diagnostic criteria for extrapulmonary tuberculosis are specific to each location, by identifying the agent in pathology, histology and cytology. Criteria for diagnostic confirmation of pulmonary tuberculosis:

POSITIVE: Two direct smear positive, a positive direct smear and culture positive, smear-positive direct and radiographic images suggestive of tuberculosis or two or more negative smear and positive culture;

NEGATIVE: two negative sputum smears with suspected radiologic and clinical findings or other tests (biopsy) allowing the physician make a diagnosis of pulmonary tuberculosis or extrapulmonary (Pleural peripheral lymphatic, osteoarticular, genitourinary, meningeal and others).

The differential diagnosis should be done with the aspiration lung abscess, pneumonia, pulmonary mycoses (paracoccidioidomycosis histoplasmosis), sarcoidosis and lung cancer, among other pathologies. The mediastinal lymph node has as the main differential diagnosis of lymphoma.

4. TREATMENT AND OTHER CONDUITS

Treatment should continue for 6 months (2 months of daily doses of rifampin, isoniazid and pyrazinamide followed by 4 months of daily doses of rifampin and isoniazid). The smear should be performed monthly after the initiation of treatment. In patients with pulmonary lesions, initially positive, proven cure will be given when, after completing the treatment, the patient has two negative sputum smears. The cure for high unproven occur if, upon completion of treatment, the patient has not undergone the smear for closure. In patients with pulmonary lesions initially negative or extrapulmonary TB, the cure for high dadaquando be completed treatment and is based on clinical and radiological criteria. It is considered treatment failure when persistent sputum positivity at the end of the correct treatment or when patients strongly positive (sputum, or + + + + +) remain so until the fourth. No monthly or initial positivity followed by negativity new and positive for two consecutive months, from April. th month of treatment. The emergence of a few bacilli (+) in the direct examination of sputum at the time of May. Or 6. First month alone does not necessarily mean failure of the scheme, and the patient must be accompanied with bacteriological tests for better definition.

Sequelae of the disease and / or treatment and may be progressive dysfunction observed in patients with severe immunosuppression or risk permanent, as in patients with AIDS, cancer, chronic renal failure, silicosis, paracoccidioidomycosis, users of glucocorticoid, chronic

tuberculosis MDR and others. Patients with diseases that interfere with the immune system, such as diabetes, gastrectomy, alcoholics, drug addicts, those who presented with prolonged evolution takes smear negativity, those who dropped out or took drugs irregularly, among others, are more likely to present sequelae and / or prolonged dysfunction.

5. PREVENTION

Surveillance of cases of work-related tuberculosis must follow the procedures indicated in the introduction to this chapter. The tuberculosis is a reportable disease and research mandatory. The specific control measures are based on educational measures and the dissemination of information, immunization, early diagnosis and appropriate treatment. Are indicated:

Monitoring of contacts:

Indicated primarily for communicating that coexist with infectious TB and adults who live with patients younger than 5 years, to identify the possible source of infection;

BCG VACCINATION: children aged 0-4 years, with revaccination at school age. Vaccinating health workers not reactive to the tuberculin skin test;

Chemoprophylaxis:

recommended for contacts of infectious TB, aged 5 years and not vaccinated with BCG and reactors to the tuberculin test, radiological examination with normal and no clinical symptoms compatible with tuberculosis; people infected by the bacillus (secondary prophylaxis) or not (primary prophylaxis) at a dosage of 10 mg / kg / day (400 mg) of isoniazid for a period of 6 months. Newborn of cohabiting bacilliferous: is administered chemoprophylaxis for 3 months and after that period, it is the PPD. If it is reactive, isoniazid remains until 6 months, if not the reactor, the drug is suspended and applies the BCG vaccine, tuberculin recent; HIV positive in the following cases: contacts of infectious TB, minor 5 years, household or institutional contacts of patients with active tuberculosis, regardless of tuberculin skin test; reactors to PPD (5 mm or more) and

asymptomatic, not reactive to PPD (induration less than 5mm), with CD4 counts below 350 cells/mm³ or less than 1,000 total lymphocyte cells/mm³; patients with radiological lesions or scars with the documentary record to have been reactive to PPD. Household contacts of smear positive and immunosuppressed by drugs or immunosuppressive

diseases, under careful medical decision;

HEALTH EDUCATION:

clarification regarding aspects of the disease, its transmission, prevention and treatment. It is recommended to verify the adoption, by the employer, taking measures to control occupational risk factors and health monitoring identified in the PPRA (NR 9) and PCMSO (NR 7), facilities to meet the standards Universal precautions, and other regulations - health and environmental - in the states and municipalities. The periodic health of workers exposed part of PCMSO should include standardized protocols aimed at early detection of disease and, if necessary, to search for bacilli in sputum

Sputum and skin test (PPD).

In rural areas, should be done with sanitary control of livestock vaccination of animals and, if necessary, elimination of infected cattle and tuberculin-positive, and the sanitary inspection of products, especially milk, ensuring its proper pasteurization.

Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the SUS, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT for Social Security

As described in Chapter 5;

- direct the employer to adopt the technical and managerial procedures to eliminate.

6.3.2 ANTHRAX (Anthrax) ICD-10 A22. -

1. DEFINITION OF THE DISEASE - DESCRIPTION

Zoonosis caused by *Bacillus anthracis*, gram-positive organism, manifesting itself in humans,

In three clinical forms: cutaneous, gastrointestinal and lung. Meningitis and septicemia may be complications of all these forms.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The disease has worldwide distribution and occurs in isolated cases during the year, occasionally in the form of epidemics. Stems from human exposure to the bacillus in industrial activities, crafts, agriculture Oueme laboratories, and is therefore associated with the work, for example, by direct contact of people with hair sheep, wool, leather, skin

and bones, special animals from Africa and Asia. In agricultural activities, occurs at the contact of man with pig, horse sick or shares, derivatives and products of infected animals.

The main risk groups are the keepers of animals, farmers, workers in slaughterhouses, tanneries, bone crushing, shearing sheep, raw wool handlers, veterinarians and their assistants. For its rarity and specificity almost certain workers, can be considered an occupational disease or work related disease, the Group I of the Classification of Schilling.

3. CLINICAL AND DIAGNOSTIC

In man, the gateway is the most common skin in 90% of cases, with formation of necrotic black scab that can lead to a cure or a septicemia, through the lymphatic vessels, leading to death. The respiratory form, ordisease of the cutters of wool, is associated with aspiration of material contaminated byB. Anthracis, triggering an extensive pneumonia that progresses to septicemia and death. It begins with malaise, asthenia, myalgia, moderately high body temperature, non-productive cough and, rarely, tightness of the chest. Contamination by ingestion causes gastrointestinal form, which is manifested by nausea, vomiting, anorexia and fever followed by abdominal pain, hematemesis and sometimes dysentery. It can progress to sepsis, shock and death.

The ingestion of contaminated food has been associated also with the oropharyngeal anthrax andpharynx.

Themeningitis by anthrax can complicate skin disorders, pulmonary or gastrointestinal, although this occurs in less than 5% of patients. The onset of symptoms meningeana coincides with the occurrence of the primary lesion or shortly thereafter. The main symptom is characterized by hemorrhagic meningitis, with death arising from one to six days after onset. They have also been reported encephalomyelitis and cortical hemorrhage. The meningoenkephalitis, very rare, also has a fatal outcome.

The diagnosis can be confirmed by Gram-positive B. anthracis whelk fluids, pleural or cerebrospinal fluid. Serological diagnosis is given by the exams with ELISA (enzyme immunoassay) and western blot.

4. TREATMENT AND OTHER CONDUITS

In cutaneous forms, the drug of choice is penicillin. In mild forms, using penicillin V (250 mg, VO, 6 / 6 hours for 7 days). Extensive forms, administer penicillin G procaine, (300,000 to 600,000 units, IM, 12/12 hours, 7 days). Other drugs are available for use tetracycline and erythromycin. Excision of skin lesions is not indicated. The forms of

pulmonary, gastrointestinal and meningeanas should be treated based on

Extrapolation from animal experiments with crystalline penicillin G (4 million units IV every 4-6 hours, 7-10 days). (The supportive treatment should be carried out according to need, for example, volume infusion, vasopressor drugs, oxygen, etc.). The neck swelling may require tracheostomy.

Hospitalized patients should remain in isolation restricted.

5. PREVENTION

Surveillance of cases of carbuncle (anthrax)work-related must follow the procedures indicated in the introduction to this chapter. The awareness of workers about the risks involved in handling potentially contaminated materials and procedures to be adopted for prevention are essential.

It is recommended:

- regular and effective cleaning equipment and work areas and facilities for personal hygiene of employees;
- decontamination of raw materials and disinfection of potentially contaminated animal products with hypochlorite or formaldehyde;
- vaccination of workers in industries with high risk of contamination by anthrax;
- reporting to health authorities of all confirmed cases of anthrax and surveillance of exposed *B. anthracis* for 7 days, the maximum incubation period of anthrax;
- use of chemoprophylaxis after occupational exposure to aerosols *B. anthracis*, using the same treatment regimen for cases of inhalation;
- indication of antimicrobial prophylaxis after ingestion of contaminated food or by injection of virulent bacilli through the skin, with intramuscular penicillin, as recommended for extensive skin lesions, and surveillance for 10 days;
- appropriate use of PPE.

Theanthrax agriculture should be controlled by vaccination of animals in endemic areas and proper handling of the carcass. Contaminated food and fertilizer should not be used.

The diagnosis should be performed on all animals suspected of having died of anthrax. Infected animals and the dead must be destroyed quickly, preferably by burning. It is recommended to check the suitability and adoption by the employer of the control measures of occupational risk factors and surveillancehealth identified in the PPRA (NR 9) and PCMSO (NR 7), and other regulations - environmental and health-existing in states

and municipalities. Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the information systems of the SUS, the DRT /

MTE and the union.

Provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;

•direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

BRUCELLOSIS ICD-10 A23. -

1. DEFINITION OF THE DISEASE - DESCRIPTION

It is primarily a zoonosis of domestic and wild animals. It is caused by bacteria *Brucella melitensis*, *B. abortus*, *B.* and *B. suis canis*. The man contracted the disease through contact with sick animals, its carcass, blood, urine, vaginal discharges, aborted fetuses, placenta or by ingestion of milk or dairy products from

Of infected animals. It can also occur through contamination of laboratory accidents. Transmission from person to person has been suspected in some special situations, but seems to be extremely rare.

The incubation period is highly variable, ranging from 5 to 60 days, even months. Generally, the onset of symptoms occurs two to three weeks after exposure.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The disease occurs due to occupational exposure to *Brucella melitensis*, *B. abortus*, *B. suis*, *B. canis* in slaughterhouses, packing plants, meat or handling products, milk and dairy product manufacturing and related activities.

For its rarity and the specificity that has in certain types of work activities, the Brucellosis can be considered as an occupational disease or work related disease, the Group I of the Classification of Schilling.

3. CLINICAL AND DIAGNOSTIC

The brucellosis-disease can manifest as acute, subacute or chronic fever syndrome, that in cases of long duration may take the characteristic undulating, with malaise, easy fatigue, arthralgia, myalgia, pain in the calves and lower back, headache, listlessness and depression. Lymphadenopathy can be observed very expressive and rarely

hepatosplenomegaly. In acute, the disease duration is up to two months in subacute lies between two months and one year, and chronic exceeds this limit. Many patients may have limited changes to the system as a body and bones and joints (sacroiliitis, osteomyelitis, paraspinal abscess), liver and gallbladder (hepatitis, cholecystitis), digestive tract (acute ileitis, colitis), urinary tract (pyelonephritis, glomerulonephritis, renal abscess) and respiratory (pneumonia, pleurisy, lung solitary), heart and great vessels (endocarditis, pericarditis), nervous system (asthenia, depresssão, meningitis, encephalitis, radiculoneuritis, myelitis, peripheral neuropathy, cerebral mycotic aneurysm) skin and soft tissue (rashes, ulcers, vasculitis).

Laboratory diagnosis is accomplished through:

- Isolation Brucella in culture of blood, bone marrow, or other secretions of tissue fragment;

- agglutination test with titers greater than or equal to 1 / 160 or 4-fold increase in the titers of agglutination test in serial tests, 2 to 3 weeks between them (70 to 10 days after infection, specific IgM can be detected for Brucella

The differential diagnosis must be made with the diseases that behave as fever of unknown origin (Tuberculosis, lymphoma, abscesses, toxoplasmosis, infectious mononucleosis, rheumatoid arthritis, among others), with endocarditis

Bacterial and typhoid fever.

4. TREATMENT AND OTHER CONDUITS

Treatment is done with:

- doxycycline or minocycline (100 mg, PO, 12.12 hours for 45 days) associated with rifampicin (600 - 900mg/dia, PO, once daily for 45 days).

The alternatives available are:

- doxycycline or minocycline (100 mg, PO, 12.12 hours for 45 days) associated with streptomycin (1 g daily IM for 3 weeks) or gentamicin (5 mg / kg / day IM or IV, divided into equal portions of 8 / 8 hours);

- sulfamethoxazole (800) / trimethoprim (160), 12/12 hour, VO, for six weeks, associated with gentamicin (5mg/kg/day, IM or IV, divided into equal portions of 8 / 8 hours).

Relapses should be treated with the same antibiotic regimen. Relapses generally are not due to antibiotic resistance, but the kidnapping of agents by an organ that prevents effective action of the drug.

Doxycycline should not be used on children under the age of seven

years or in pregnant women after the sixth month of pregnancy.

5. PREVENTION

Surveillance of cases of work-related brucellosis must follow the procedures indicated in the introduction to this chapter. The health agencies should act in a manner coordinated with the agencies responsible for control of livestock, which can alert health monitoring and preventing the distribution and consumption of infected, to reduce morbidity and mortality of the disease. Although the Brucellosis not notifiable disease in the national territory may be in some states or municipalities. In the presence of outbreaks must be notified to intensify control measures indicated:

HEALTH EDUCATION:

Informing the public about the benefits of consuming milk and dairy products pasteurized properly, educating the workers who care for animals on the risks of disease and care to avoid contact with sick birds or potentially contaminated;

ANIMAL CONTROL: perform serologic tests and dispose of infected animals;

CONTROL PRODUCTS:

Those derived from animal sources require appropriate health surveillance for milk and its derivatives; care in the handling of placentas, fetuses and animal secretions. Provision must be made to disinfect contaminated areas;

MANAGEMENT OF PATIENT:

Precautions with drainage material and secretions. Disinfection should be performed concurrent purulent secretions and investigation of contacts for treatment, control and adoption of preventive measures. In epidemic situations, investigate common sources of contamination, which generally are derived from milk and unpasteurized. Confiscate suspected food until they are established measures to prevent outright;

IN CASE OF WORKERS EXPOSED: should be observed biosecurity measures, provided the right PPE and facilities for personal hygiene.

In some cases it may be necessary to control the infection in domestic animals (dogs, cattle), done by vaccines, serological tests for early diagnosis, chemotherapy and, if necessary, sacrifice of the infected animal. It is recommended to check the suitability and compliance by the employer, the control measures of occupational risk factors and health

promotion identified in the PPRA (NR 9) and PCMSO (NR 7), and other regulations - environmental and health - in the states and municipalities.

Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the information systems of the SUS, the DRT / MTE and the labor union

LEPTOSPIROSIS ICD-10 A27. -

1. DEFINITION OF THE DISEASE - DESCRIPTION

Ubiquitous zoonosis caused by pathogenic spirochetes of the group Leptospiracea. The clinical presentation is variable, with asymptomatic or mild to severe, manifested by jaundice, bleeding, anemia, kidney failure, liver involvement and meningitis. Recovery is usually complete in 3-6 weeks. The severity of infection depends on the infecting dose, the variety of serological *Leptospira* and the patient's condition. The incubation period varies from 3 to 13 days, reaching up to 24 days.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The leptospirosis Zoonoses are true. Rodents are the main reservoirs of the disease, especially the domestic ones. Act as carriers cattle, sheep and goats. The transmission is accomplished by contact with water or soil contaminated with urine of carrier animals, more rarely by direct contact with blood, tissue, organ and urine of these animals. No inter-human transmission, except for the intrauterine fetus.

The Leptospirosis work-related has been described in workers who perform activities in direct contact with water or in locations with contaminated animal waste carriers of germs, as the work performed in mines, tunnels and sewage into watercourses and drainage; contact with rodents or domestic animals; preparation of food of animal origin, in fish, dairy and other similar activities.

In some workers, Leptospirosis can be considered a work related disease, the Group II of the Schilling classification, since the circumstances of occupational exposure to *Leptospira* can be considered as contributors in the set of factors associated with the etiology of this infectious disease.

3. CLINICAL AND DIAGNOSTIC

The clinical manifestations of Leptospirosis exhibit variable spectrum: from asymptomatic to oligosymptomatic (Anicteric), flu syndrome simulating until severe icteric forms with hepatorenal involvement and acute renal failure. After an incubation period of 7 to 10 days and ranging from two days to more than one month, the disease appears. The anicteric form affects 60 to 70% of cases and has two phases:

Septicemia: characterized by hepatomegaly and, more rarely, splenomegaly, gastrointestinal hemorrhage, myalgia involving calves (mostly), thigh, abdomen and paraspinal muscles, photophobia, chest pain, dry cough with or without hemoptysis, rash macular, maculopapular, or urticarial petechiae, mucosal hyperemia lasting 4-7 days;

IMMUNE: when there is severe headache, vomiting and signs of meningeal irritation, uveitis, lasting 1-3 weeks.

The icteric form, also called Weil's disease, progresses to renal failure, hemorrhagic phenomena and hemodynamic changes. Symptoms are more intense than in the anicteric form, lasting 1-3 weeks, with mortality rates 50 to 20%. Laboratory tests for diagnosis are the culture of blood or cerebrospinal fluid (the first week and beginning of the second week of illness) or urine (after the second week there) and serological reactions: reaction of macroscopic and microscopic agglutination test, complement fixation reaction, hemagglutination reaction, ELISA and others.

It is considered as a confirmed case who fills any of the following criteria:

- isolation *Leptospira* any clinical specimen;
- associated with clinical symptoms suggestive serologic conversion, ie 4-fold increase or more in the title obtained by the microscopic agglutination reaction between the acute and convalescent;
- specific IgM by ELISA.

The differential diagnosis must be made with:

Anicteric FORM: influenza, typhoid fever, septicemia by gram-negative, dengue, acute appendicitis, acute cholecystitis, malaria, acute pyelonephritis, toxoplasmosis;

FORM jaundice: icteric forms of typhoid fever, sepsis by Gram-negative, yellow fever, hepatitis, malaria *P. falciparum*, among others.

4. TREATMENT AND OTHER CONDUITS

For adults, it is recommended crystalline penicillin G in doses of 1,

5 million units, IV, 6 / 6 hours for 7 to 10 days or tetracycline, 2 g daily, orally, for 6 / 6 hours, even before the fifth day of illness. After the fifth day, the medication does not alter the course of the disease. The allergic to penicillin or tetracycline can use ceftriaxone. If necessary, can be used as supportive measures oxygen and intravenous fluid replacement.

In patients who develop renal failure is indicated the installation of early peritoneal dialysis, the first signs of oliguria, which lowers the rate of mortality of the disease.

5. PREVENTION

Surveillance of cases of Leptospirosis work-related must follow the procedures indicated in the introduction to this chapter. The Leptospirosis is not a national reportable disease, and may be so in some states and municipalities. In the presence of outbreaks must be notified to that adopt control measures indicated. Among the measures of prevention and control are:

- epidemiological surveillance, particularly early in the period of heavy rains in areas of cyclical occurrence;
- early and adequate treatment of critically ill patients seeking to reduce the lethality of the disease;
- Equipment adequate protection for workers whose activities in wetlands,
 - Sewers, rivers, ponds, silos, warehouses;
- measures of anti-ratização and rodent control, improvement of sanitary conditions of the population, food protection;
- proper disposal of leftover food and garbage in general;
- orientation of the population about the risks of increased disease in the rain and floods, and on preventive care, avoiding flooded areas without personal protection;
- orientation of workers who are at risk, beyond the provision of individual protection equipment and adequate facilities for personal hygiene.

It is recommended to check the suitability and compliance by the employer, the control measures of occupational risk factors and health promotion identified in the PPRA (NR 9) and PCMSO (NR 7), and other regulations - environmental and health - in the states and municipalities. Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the information systems of the SUS, the DRT /

MTE and the labor union;

- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control of risk factors.

TETANUS 6.3.5 ICD-10 A35. -

1. DEFINITION OF THE DISEASE - DESCRIPTION

Acute disease produced by the potent neurotoxin (tetanospasmin) of *Clostridium tetani*. The tetanus toxin prevents the inhibition of the reflex arc of the spinal cord, promoting tonic excitatory reflexes typical in many regions of the body.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The *C. tetani* is an anaerobic bacillus, found in nature in widely distributed in the form of spores in soil, especially when treated with animal manure, on thorns of bushes and small tree branches, rotting in water, in dirty rusty nails in instruments work or cans contaminated with dust or dirt from the street, in human or animal feces in wires catgut and injection needles not properly sterilized.

It is spread by feces of horses and other animals and infects humans when the spores enter through contaminated injuries in general are of a piercing, but also of lacerations, burns, umbilical stump is not treated properly, etc.. The presence of necrotic tissue, pus or foreign location facilitates the reproduction of the bacillus, which is not invasive and acts at a distance by its toxin.

Occupational exposure of workers is relatively common and takes place mainly in industrial accidents (agriculture, construction, mining, sanitation and garbage collection) or commuting accidents. The disease in employees arising from accidents at work might be considered a work related disease, the Group I of the Classification of Schilling.

3. CLINICAL AND DIAGNOSTIC

The incubation period varies from 40 to 50 days, usually 7 days. The shorter the incubation time, with more rapid disease progression and greater severity. The clinical manifest, sequentially, for localized symptoms, with mild spasms in the region of injury; premonitory symptoms such as irritability, tingling, aching backs and shoulders and permanent contracture (muscle stiffness), which may affect muscle groups located in or present generalized hypertension (more common) and paroxysmal spasms or contractures. The diagnosis is established by research on outbreak of suspected bacillus by direct smear, culture or

inoculation in anaerobic material in guinea pig and focus of observation for 8 days. Generally have poor outcomes. The diagnosis is mainly clinical.

The differential diagnosis must be made with other causes of trismus and tetany, anger, hysteria, intoxication strychnine syndrome of rigidity, as Parkinson syndrome.

4. TREATMENT AND OTHER CONDUITS

The patient toxoid, particularly in more severe forms, should be preferably treated in the intensive care unit, and therapeutic measures that prevent or control the complications (respiratory, infectious, cardiovascular, metabolic), which may lead the patient to death.

Therapeutic measures include:

- admission to the silent room, in darkness, with a maximum reduction of auditory stimuli, visual, tactile, and others;
- supportive medication: sedatives (benzodiazepines), muscle relaxants, analgesics (sometimes powerful) airway maintenance free, and may require mechanical ventilation, respiratory therapy;
- tetanus serum (SAT), after sensitivity testing at a dose of 20,000 IU in 100 ml NaCl 0.9% and infused IV for 1 hour, or gamma globulin (IGATE) 3000-6000 IU distributed in one or two muscle masses ;
- antibiotics using penicillin G 2 million units IV every 6 hours, or tetracycline, 500 mg IV every 6 hours for 10 days, or even, metronidazole 500 mg IV, 6 / 6 hours of 7 to 10 days;
- debridement and cleaning of suspected outbreaks.

At the time of discharge, should be applied to tetanus toxoid vaccine in muscle mass than the one applied anti-tetanus serum.

With regard to dysfunction or disability, overcome the acute stage, when severe, may result in permanent sequelae, particularly neurological disorders, which should be evaluated as to the nature, location, involvement of social and labor activity.

5. PREVENTION

Surveillance of cases of tetanus-related work must follow the procedures indicated in the introduction to this chapter. The tetanus is a disease of compulsory notification and investigation required in the national territory.

Among the classical measures of prevention and control are:

VACCINATION: maintaining adequate levels of immunization coverage of the population, particularly children, elderly, people with chronic ulcers on the legs and foot ulcerations caused by leprosy. All

workers employed in high-risk activities, such as farmers and construction workers, sanitation workers and garbage collection and mining should be vaccinated;

PROPHYLAXIS: regarding the need for active and passive immunization in patients with suspicious injuries, you should evaluate the history of previous immunization and type of injury. Outbreaks are, potentially, the bacillus of contamination: injuries of any nature contaminated by dust, dirt, animal feces or human; fractures with torn tissue and foreign bodies, burns, bites of poisonous animals, dogs, wild animals and cat scratches. Any suspected injury should be cleaned with soap and water, besides being widely debrided. It should be emphasized that the use of benzathine penicillin in the prophylaxis of tetanus, is not effective.

It is recommended to check the suitability and compliance by the employer, the control measures of occupational risk factors and health promotion identified in the PPRA (NR nine) and PCMSO (NR 7), and other regulations - health and environmental - existing in states and municipalities.

Exposed workers should be guaranteed:

- adequate working conditions;
- guidance on the risk and prevention measures;
- vaccination;
- facilities for personal hygiene (showers, sinks);
- personal protective equipment (clean clothes, gloves, boots, head protection, etc.

6.3.6 psittacosis, ornithosis, GROOMS POULTRY DISEASE
ICD-10 A70. -

1. DEFINITION OF THE DISEASE - DESCRIPTION

The psittacosis or ornithosis is an acute infectious disease produced by chlamydia (*C. psittaci* and *C. pneumoniae*).

The disease usually is mild or moderate and may be severe in elderly untreated. The incubation period ranges from one to four weeks and the period of communicability lasts weeks or months.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The most common sources of infection *C. psittaci* are parakeets, parrots, pigeons, ducks, turkeys, canaries, among others, which transmit the infection through their feces dried and scattered with the dust being

sucked in by the patients. Although rare, can be transmitted via respiratory, person to person, in the acute phase of illness. It is a zoonosis that affects workers of poultry breeding, veterinary clinics, zoos and biological laboratories.

The *C. pneumoniae* infects only humans and is transmitted from person to person.

For its relative rarity and specificity, Psittacosis / ornithosis can be considered as an occupational disease or work related disease, the Group I of the Classification of Schilling, the workers of poultry farms and poultry farms (ducks, geese, parakeets, pigeons, etc..) employees of trading houses such animals, veterinary , foresters and others in confirming that the circumstances of occupational exposure.

3. CLINICAL AND DIAGNOSTIC

The disease's incubation period is from one to four weeks and its transmissibility can last weeks or months. Both chlamydia can cause similar clinical outcomes. The infection can be subclinical, with self-limiting episodes similar to the flu, with headache, fever syndrome, prostration, chills, muscle pain, bloating abdomen, constipation or diarrhea, to pneumonia clinical course characterized by acute, often with extrapulmonary manifestations, and delirium, roseoliformes skin lesions similar to those of typhoid fever, epistaxis, splenomegaly, accompanied by impairment of upper or lower airways, causing pneumonia, bronchitis, pharyngitis, otitis media and sinusitis. It is rare to observe such complications pericarditis, myocarditis, endocarditis, superficial thrombophlebitis, hepatitis, encephalopathy. The pulmonary condition is compatible with atypical pneumonia with little cough or sputum purulent. The pulse is slow and its gradual increase, with tachypnea, is bad prognosis. The outcome may be favorable (with prolonged convalescence) or severe, with a mortality that reaches 30%.

Laboratory diagnosis is based on the increase in four-fold increase in the reaction of complement fixation between acute and convalescent phases, obtained with an interval of two to three weeks between each collection. In the presence of suggestive clinical picture, may be considered as evidence of infection.

The isolation of the agent in the blood or secretions, and tissue culture, although possible, it is difficult to implement, requiring specialized laboratories to perform it.

The differential diagnosis must be made with acute viral pneumonia, caused by rickettsia or mycoplasma and culture-negative endocarditis. If there are skin changes should be made differential

diagnosis of typhoid fever.

4. TREATMENT AND OTHER CONDUITS

In adults, tetracycline, 500 mg daily of 6 / 6 hours or doxycycline 100 mg daily of 12.12 hours for 14 to 21 days. In children younger than 7 years should be used erythromycin 30-40 mg / kg / day PO for 6 / 6 hours.

The improvement occurs in 48 to 72 hours after initiation of therapy, although it may be a little more relapses occur lenta.Podem that should be treated the same as the primary infection.

5. PREVENTION

Surveillance of cases of psittacosis and ornithosis-related work must follow the procedures indicated in the introduction to this chapter. There is no vaccine available and no specific actions are developed for disease surveillance in the health services.

The cases should be diagnosed and treated early for prevent complications and disease transmission. In general it is not reportable disease, and may be so in some states or municipalities.

Main control measures are:

GENERAL: health education to alert the population risk of exposure to the reservoirs, the need to regulate the import, breeding and transport of birds, use of antibiotics or quarantine these animals, when indicated, monitoring of places of sale of livestock, poultry, poultry . The birds suspected source of infection for humans should be evaluated by a veterinarian and disposed of in case of infection;

SPECIAL: concurrent disinfection of all secretions. Clean terminal;

DISPOSAL OF THE SOURCES OF INFECTION: sites with infected poultry, we can eliminate them or treat them and make local disinfection;

INVESTIGATION OF CONTACT: identify the origin of the infected birds. The body of the affected animal should be subjected to disinfection before discharge. People exposed to infection should be observed for development of fever or other symptoms. Exposed workers should be guaranteed:

- adequate working conditions;
- guidance on the risk and prevention measures;
- facilities for personal hygiene (showers, sinks);
- appropriate individual protective equipment (clean clothes, gloves, boots, head protection).

It is recommended to check the suitability and compliance by the

employer, the control measures of occupational risk factors and health promotion identified in the PPRA (NR 9) and PCMSO (NR 7), and other regulations - environmental and health - in the states and municipalities. Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the information systems of the SUS, the DRT /

MTE and the labor union;

DENGUE (dengue fever) ICD-10 A90. -

1. DEFINITION OF THE DISEASE - DESCRIPTION

Acute febrile disease, endemic-epidemic caused by one of Flavivirus of dengue (Family Togaviridae) four serological types (1, 2, 3 and 4). Humans are reservoirs and transmission occurs through the bite of mosquitoes *Aedes aegypti*, *A. albopictus* and *A. scutellaris*. After feeding of infected blood, the mosquito will be able to transmit the virus after 8 to 12 days extrinsic incubation. The mechanical transmission is also possible, when the meal is interrupted and the mosquito immediately feeds on a susceptible host next. There is transmission by direct contact of a patient or their secretions to a healthy person, nor by sources of water or food.

The disease's incubation period is 3 to 15 days, averaging 5 to 6 days. The period of communicability occurs during the period of viremia, which begins one day before the fever until the sixth day of illness. When the causative agent is known, the full name of the disease is dengue virus or dengue type 1 virus type 2, etc..

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Dengue can be regarded as a work related disease, the Group II of the Schilling classification, since the circumstances of occupational exposure to mosquitoes (*Aedes*) and / or infectious agents (Flavivirus) can be considered as risk factors in the whole factors associated with the etiology of this infectious disease.

Dengue-related work has been described in workers who perform activities in areas

Endemic in public health services and in research laboratories, and other activities in which occupational exposure can be identified.

3. CLINICAL AND DIAGNOSTIC

Manifested by abrupt onset of fever (39 ° - 40 ° C), severe

headache, pain retroocular, myalgia, arthralgia, gastrointestinal symptoms (vomiting, anorexia). Can arise in a morbilliform centrifugal 3. Or 4. Day of illness and sometimes haemorrhagic discrete (epistaxis, petechiae). There may be hepatomegaly and micropoliadenopatia. The fever usually remits within 6 days.

Laboratory tests used:

- CBC: leukopenia with lymphocytopenia;
- platelets: normal or slightly reduced;
- virological methods (up to 6. day) cultures, enzyme immunoassays, radioimmunoassays or PCR;
- serological methods: a MAC-ELISA (IgM capture) requires a single sample and is the best test for monitoring epidemiology.

The possibility of dengue hemorrhagic (Hemorrhagic fever and dengue shock syndrome) is feared. The WHO classifies dengue hemorrhagic four degrees of severity, locating in the first two more benign forms (with hemorrhagic fever) and severe in the last two (with circulatory failure), as follows:

GRADE I: fever and nonspecific symptoms, with the only hemorrhagic manifestation of the tourniquet test positive

LEVEL II: presence of spontaneous hemorrhagic phenomena;

LEVEL III: circulatory failure manifested by rapid and weak pulse, decreased pulse pressure 20 mmHg, hypotension, and cold clammy skin, restlessness;

LEVEL IV: profound shock characterized by absence of pulse and blood pressure. The less frequent clinical manifestations of dengue include disorders that affect the nervous system such as encephalitis and polyneuropathy (syndromes Reye and Guillain-concubine). These conditions may arise in the course of the disease or convalescence. Paintings have been described for hepatitis with jaundice and significant elevation of serum transaminases. The deficiencies or disorders are evaluated according to the nature of the complication or sequel, and is not specific to the dengue. The differential diagnosis must be made with influenza, rubella, measles, yellow fever, leptospirosis, infectious hepatitis and other hemorrhagic fevers.

4. TREATMENT AND OTHER CONDUITS

The treatment of classic dengue on an outpatient basis and consists of observation and use of rescue medications (do not use aspirin). The severe bleeding and shock should be treated in special environment in intensive care units, and require fluid replacement and approach of the syndrome of disseminated intravascular coagulation.

5 PREVENTION

Surveillance of cases of dengue-related work must follow the procedures indicated in the introduction to this chapter. Surveillance activities are aimed at controlling the occurrence of the disease by combating mosquito, environmental sanitation activities, orientation to reduce the breeding population of larvae *A. aegypti* (Flower pots, puddles, containers, tires, etc..) And chemical control by use of insecticides on infested areas. It is a disease of compulsory notification and investigation required, especially when it comes to the first cases of classic dengue diagnosed in one area or is suspected dengue haemorrhagic fever.

Exposed workers should be guaranteed:

- adequate working conditions;
- guidance on the risk and prevention measures;
- facilities for personal hygiene (showers, sinks);
- Adequate PPE (clean clothes, gloves, boots, head protection, etc.).

It is recommended to check the suitability and compliance by the employer, the control measures of occupational risk factors and health promotion identified in the PPRA (NR 9) and PCMSO (NR 7), and other regulations - environmental and health - in the states and municipalities.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the information systems of the SUS, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

YELLOW FEVER ICD-10 A95 .-

1 DEFINITION OF DISEASE - DESCRIPTION

Acute febrile disease caused by Flavivirus yellow fever (family Togaviridae), with clinical symptoms vary from inapparent forms to the serious and fatal. The transmission is through the bite of infected mosquitoes *A. aegypti* in Yellow Fever (FAU) and *Haemagogus* in yellow fever (FAS). The incubation period is 3-6 days after the bite of an infected mosquito, and the period of transmissibility is 24 to 48 hours

before symptoms appear 3 to 5 days.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The yellow fever persists in South America just as enzootic disease of monkeys, and by transmitting mosquitoes of the genus *Haemagogus* and *Aedes*. Human cases, few, focus among people who work or have contact with the forests. The yellow fever urban had the man as one tank and the *A. aegypti* as transmitter and South America Other workers potentially exposed accidentally include performing public health activities and working in research laboratories, farmers, forest workers in logging, and in areas affected regions.

For its rarity and because of its relative specificity, yellow fever in some workers may be considered as an occupational disease or work related disease, the Group I of the Classification of Schilling.

3. CLINICAL AND DIAGNOSTIC

The clinical picture varies from benign, non-specific, even fulminant disease characterized by multiple organ failure, particularly for bleeding. The severe form starts abruptly with the so-called period of infection, characterized by fever, chills, severe headache, lumbosacral pain, generalized myalgia, anorexia, nausea, vomiting and bleeding gums or epistaxis of low intensity.

It lasts three days, followed by periods of remission, with improvement lasting 24 hours. In severe cases, the symptoms reappear, characterizing the period of intoxication (most severe).

Laboratory tests show:

- CBC: leukopenia;
- liver tests and coagulation tests changed;
- ECG changes in ST-T;
- virological tests (up to 4. th);
- serological tests.

Regarding differential diagnosis, the mild and moderate forms are difficult to distinguish in relation to febrile diseases. The classical or serious fulminant hepatitis should be differentiated from severe fulminating, leptospirosis, malaria *P. falciparum* malaria, dengue haemorrhagic fever and septicemia.

4. TREATMENT AND OTHER CONDUITS

The classic frames and / or require hospitalization for fulminant

adoption of supportive symptomatic treatment, according to the manifestations and progression.

5. PREVENTION

Surveillance of cases of yellow fever work-related must follow the procedures indicated in the introduction to this chapter. The monitoring aims to prevent the redevelopment of the disease and maintain yellow fever under control. It is international reportable disease (must be reported immediately by the quickest route, to health authorities) and which requires epidemiological investigation of all cases. In viral hepatitis B the virus is found in all secretions and excretions of the body, but apparently, only blood, semen and saliva are capable of transmitting it. The infection is acquired, in general, during transfusions, percutaneous injections with blood products or use of contaminated needles and syringes, or even by sex, male homosexual or heterosexual. In health care workers, the prevalence of HBV is 2-4 times higher and the annual incidence is 5 to 10 times higher than in the general population.

In Viral Hepatitis C seroprevalence among healthcare workers appears to be similar to the general population.

Seroconversion of workers who suffer such accidents with contaminated material occurs in 1, 2 and 10% of workers. It is estimated that 2% of cases are due to occupational exposure. The viral hepatitis D is endemic in Western Amazonia, where, in association with the virus hepatitis B, is the causative agent of the callblack fever Lábrea, fulminante. Portanto of evolution in some workers, Viral hepatitis can be considered work-related diseases, the Group II of the Schilling classification, since the circumstances of occupational exposure to viruses can be considered as risk factors in the set of risk factors associated with the etiology of this infectious disease.

3. CLINICAL AND DIAGNOSTIC

It is characterized, in the prodromal phase, by a sudden onset of low fever, anorexia, nausea and sometimes vomiting and diarrhea. There may be headache, malaise, weakness and fatigue, and pain in hypochondrium law. The weight in the prodromal phase may be asymptomatic. In the icteric phase, the prodromal symptoms and decrease arises jaundice, hepatosplenomegaly

Painful and discreet. In the convalescent phase, the jaundice disappears with complete recovery within weeks.

The Hepatitis B and C may progress to chronicity, with or without complications. The Hepatitis B can evolve from an acute fulminant,

especially in the presence of co-infection or superinfection by virus hepatitis D.

The diagnosis is based on:

- elevation of at least 10 times normal transaminases (AST and ALT) and aminotransferases (ALT and AST);
- elevation of bilirubin;
- serological tests for identification of antigens and antibodies;
- liver biopsy (when necessary).

The prodromal phase or pre-icteric generally lasts 30 to 10 days. The icteric phase may last from a few days to several weeks, although the transaminases may remain elevated for prolonged periods 1-2 years, without necessarily indicate that infection has chronified. From the evolutionary point of view, each type of viral hepatitis have different clinical course, dependent on the viral strain virulence and immune response of each individual. In benign acute hepatitis, the trend is for healing. The evolution to chronicity, with or without complications, does not occur in HAV and HEV. The diagnosis is essentially histological chronicity. You can not define it only by clinical manifestations or disease by the time elapsed. The polyphase evolution (recrudescence) are common in HAV, while prolonged acute forms are found with high frequency in HCV and HAV with some frequency in both with good prognosis. The high clinic is a function of complete remission of symptoms, except a vague digestive symptoms and certain malaise that can persist, total or near total disappearance of jaundice, normalization of bilirubin and hepatic synthesis of evidence (prothrombin time and dosage protein), normalization of transaminases.

4. TREATMENT AND OTHER CONDUITS

In the case of acute infection, treatment is only symptomatic. The rest is relative, being dictated by the patient. The diet also depends on the desire and patient symptomatology. For chronic cases of HBV infection, the available therapy is interferon alfa-2b or lamivudine. The CHC requires use of the combination of interferon alfa-2b with ribavirin. It is not yet an ideal treatment because of its complexity.

PREVENTION

Case surveillance viral hepatitis related to work must follow the procedures indicated in the introduction to this chapter.

Preventive measures and control for HAV and HEV can be summarized in:

- sanitation, especially adequate control of water quality for human consumption and system for collecting human waste;
- educational and basic information on hygiene and ways of transmission of the disease, to prevent new cases;
- adoption of enteric isolation of the patient at home, aiming at protecting the family;
- epidemiological investigation to identify the source of contamination and adoption of preventive measures such as chlorination of water, food protection, among others;
- guidance and supervision of health professionals about the need to obey the Guidelines on Biosecurity and vaccination for the virus (there is no vaccine for dengue virus E);
- use of immunoglobulin antiviral Hepatitis A for contacts of people with acute infection or individuals injured by biological materials, known to be contaminated with the virus.

The Hepatitis B is notifiable disease in the country. The main control measures for HBV and HDV are:

- vaccination of all susceptible individuals, regardless of age, especially for those residing or traveling to hyperendemic areas. Are priority groups for vaccination: health professionals, drug users, negative individuals using blood and blood products, prisoners, residents in psychiatric hospitals, male homosexuals and sex workers.

The basic immunization schedule is 1 ml in adults and 0.5 ml in children under the age of 11 years, in three doses: a) first b) second, thirty days after c) third after the first 6 months;

- human immunoglobulin antiviral hepatitis B, indicated in cases of newborn children of mothers with HBsAg, or sexual contacts of patients with acute infection, individuals injured by contaminated material (in these, while administering the vaccine).

For control of HCV carriers and patients should be advised to avoid spreading the virus by adopting simple measures such as:

- use condoms during sexual intercourse;
- not donate blood;
- using disposable syringes, avoiding sharing.

The blood therapy services (blood centers and blood banks) and sexually transmitted diseases

Health worker must notify patients diagnosed by them and forward them to the monitoring service

Epidemiological municipal or state, to complete the investigation and receive medical care.

Exposed workers should be guaranteed:

- adequate working conditions that enable them to follow the rules

Universal precautions;

- guidance on the risk and prevention measures;
- specific vaccination for HBV;
- facilities for personal hygiene (showers, sinks);
- Adequate PPE (clean clothes, gloves, boots, head protection,

etc.)..

It is recommended to check the suitability and compliance by the employer, the control measures of occupational risk factors and health promotion identified in the PPRA (NR 9) and PCMSO (NR 7), and other regulations - environmental and health - in the states and municipalities.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the information systems of the SUS, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;

DISEASE BY HUMAN IMMUNODEFICIENCY VIRUS (HIV)
ICD-10 and B20-B24.

1. DEFINITION OF THE DISEASE - DESCRIPTION

The disease by the human immunodeficiency virus (HIV) is a disorder of cell-mediated immunity, caused by a virus of the subfamily Lentivirinae (Family Retroviridae), characterized by opportunistic infections, malignancies (such as Kaposi's sarcoma and the non-Hodgkin lymphoma), neurological disorders and a variety of other syndromes. The Acquired Immunodeficiency Syndrome (AIDS or Aids) is the most serious manifestation of a spectrum of HIV-related conditions. The risk of people infected, untreated, develop AIDS is 1-2% per year in the first year after infection and about 5% thereafter. The cumulative risk of developing the syndrome in untreated infected is about 50%.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The transmission of HIV can occur through semen, vaginal secretions by, milk, blood and derivatives, by transfusion or by needles and syringes contaminated with blood from infected patients (in injecting drug users), by congenital in 15 to 50% of pregnancies of mothers infected by occupational accidents with contaminated needles or syringes

or other circumstances related to work.

The seroconversion rate after occupational exposure to percutaneous injury has varied between 0, 1, 0, 4%, higher depending on the inoculum size, duration of contact and extent of the injury. The international scientific literature records about 60 cases, confirmed by 1999, resulting from occupational exposure in healthcare workers, due to accidents with needles or sharps contaminated surgical material, handling, packaging or use of blood or its derivatives and contact materials from infected patients.

Thus, in certain workers, disease by the human immunodeficiency virus (HIV) can be considered a work related disease, the Group I of the Classification of Schilling, given the circumstances of occupational exposure to the virus are accidental or occur in specific conditions of work, if well documented and excluded other risk factors.

3. CLINICAL AND DIAGNOSTIC

The symptoms of HIV infection is complex but can be summarized into four groups:

GROUP 1: Acute infection - appears 3-6 weeks after infection and are manifested by fever, arthralgia, myalgia, maculopapular rash, urticaria, diarrhea and other nonspecific symptoms. Lasts up to two weeks and

Spontaneous regression;

Group two: asymptomatic infection - a period that varies in time, but lasts an average of 10 years;

Stomach cancer ICD-10 C16. -

1. DEFINITION OF THE DISEASE - DESCRIPTION

Between 90 to 95% of neoplasms of the stomach are adenocarcinomas and the remaining 50-10% are divided between leiomyosarcomas and lymphomas. Of adenocarcinomas, 75% are ulcerated, 10% were polypoid and 10% are scirrhous. As for location, 50% are located in the pylorus and antrum, 20% in the lesser curvature;

20% in the body, 7% in cardia, and 3% in the greater curvature (greater curvature ulcers are rarely benign.)

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The causes of Stomach cancer are unknown. Factors likely genetic, environmental, infectious, dietary and nutritional have been associated with disease. The gastric cancer is 3-6 times more common in patients with pernicious anemia, an entity associated with genetic

inheritance. It is more common in people of blood group A and in patients with chronic atrophic gastritis than the general population. Among the dietary habits associated with an increased risk for the disease are: high salt intake, diet high in nitrates (found in water, vegetables and meats preserved), high consumption of carbohydrates and low intake of raw vegetables, salads, fresh fruits and animal proteins.

The association of gastric cancer with infection *Helicobacter pylori*, described recently opened an explanatory perspective of high interest and high expectations. IARC formally recognizes the infection *H. Pylori* as an etiological factor of stomach cancer.

Occupational exposure to asbestos or asbestos constitutes a risk factor of occupational nature of the relatively well-documented epidemiological point of view. Cohort studies of workers exposed for long periods of work show that the incidence of Stomach cancer is 30 to 100% higher than in similar occupational groups, but without occupational exposure to asbestos. Workers in coal mines, oil refineries and rubber industry, among others, epidemiological observations are not yet conclusive, a higher incidence of stomach cancer without knowing the etiological factor.

The Stomach cancer can be classified as work related disease, the Group II Classification Schilling, in workers occupationally exposed to Asbestos is considered as a risk factor in the set of factors associated with the etiology of this multifactorial tumor.

3. CLINICAL AND DIAGNOSTIC

The gastric cancer frequently progresses to advanced stages before symptoms and signs develop. The clinical picture manifested by anorexia, feeling of fullness early aversion to meat, weight loss and abdominal pain. Vague abdominal discomfort may be initially relieved with antacids, may be followed by secondary symptoms to a mild anemia by iron deficiency; dysphagia due to lesions in the esophageal junction, vomiting due to the obstruction in gastric emptying, diarrhea secondary to plastic Linite, incarcerating the small intestine, rectal urgency and stool in the morning to tape, due to metastases.

The diagnosis of gastric cancer is based on clinical history and, in later stages, the physical exam. Patients with persistent complaints related to the gastrointestinal tract should be extensively investigated for gastric cancer with the aid of contrast studies of the upper gastrointestinal tract, endoscopy, cytology exfoliative brushed biopsy and analysis of stomach acid. This set of tests can detect more than 95% of stomach cancers.

4. TREATMENT AND OTHER CONDUITS

The line includes classic surgery with curative resection, palliative or prophylactic radiotherapy or chemotherapy. For the routing of therapeutic procedures and legal systems have been used for staging, in the case of stomach cancer, are based on the degree of penetration into the stomach wall and lymph node involvement and distant metastases. The staging system most widely used in medical oncology is defined as follows:

STAGE zero: without serosal involvement, no tumor the level of resection, no lymph node involvement. Five-year survival: 60%;

STAGE I: only one of the three above criteria is present. Survival over five years: 40%;

STAGE II: two of the above criteria are present. Five-year survival: 20%;

STAGE III: all three criteria are present. Five-year survival: less than 5%.

Indeed, in the stage of cancer, large patient populations and follow-up studies have reported a survival rate at five years less than 5% for those patients with serous or lymph node involvement. The type of gastric cancer associated with better prognosis is the superficial spreading: a five-year survival is achieved in almost 90% of patients. The type polypoid, ulcerated and plastic Linite have a progressively worse prognosis. As for the degree, tumors which show a high degree of malignancy had a poorer prognosis.

5. PREVENTION

The prevention of stomach cancer related to work based on the procedures for monitoring the environment, working conditions and the effects or damage to health, described in the introduction to this chapter. The control of exposure to asbestos and other risk factors identified may help to reduce the incidence of disease in occupational groups at risk. The environmental control measures aimed at eliminating or reducing exposure to concentration levels close to zero or within the limits established by:

- entrapment and isolation procedures of work sectors;
- use of hermetically sealed systems in the industry;
- adoption of standards of hygiene and safety with stringent exhaust ventilation systems adequate and efficient;
- systematic monitoring of ambient air concentrations;
- changes in work organization that allow reducing the number of exposed workers and exposure time;

- measures of general cleaning of work environments and facilities for personal hygiene, like bathing, washing hands, arms, face and exchange of clothing;

- provision by the employer of appropriate personal protective equipment in good repair, as indicated in a complementary way to measures of collective protection.

The procedures for health surveillance of workers exposed to asbestos are described in the protocol Mesotheliomas in this chapter.

It is recommended to check the suitability and compliance by the employer, the control measures of occupational risk factors and health promotion through the PPRE (NR 9) and PCMSO (NR 7), and other regulations - health and environmental - in the states and municipalities.

The periodic medical examination aims to identify signs and symptoms for early detection of disease.

In addition to thorough clinical examination, we recommend the use of standardized instruments and carrying out laboratory tests indicated by the nature of occupational exposure. Measures of health promotion and tobacco control should be implemented.

Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the information systems of the SUS, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

Angiosarcoma LIVER ICD-10 C22. 3

1. DEFINITION OF THE DISEASE - DESCRIPTION

The primitive cell carcinomas (primary) liver are primary hepatocellular carcinoma, called hepatoma or parenchymal cell carcinoma, accounts for about 90% of all cases of malignant liver tumors. The cholangiocarcinoma (Hepatic bile ducts) are responsible for about 5 to 7% of cases and the rest are mixed tumors. Among the rarer are the hepatoblastomas, the angiosarcomas or hemangiosarcomas (Kupfer cells or cells of the sinus) and other sarcomas.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The etiology of hepatic angiosarcoma is still unknown. The risk of occurrence increases in people exposed to arsenic, anabolic steroid, thorium dioxide (Thorotrast) and vinyl chloride monomer.

The risk factor of occupational nature most well-documented, from 1974, is occupational exposure to vinyl chloride, volatile substance used in polymerization, which results in polyvinyl chloride (PVC).

It is present in plants vinyl chloride or in the production of PVC (polymer), where there is risk of exposure to chloride vinylamonomero (VCM). The observation does not apply to industries of plastic objects, where the PVC is a raw material, solid granules and no handling of VCM. On the other hand, if the PVC undergoes pyrolysis at high temperature, the VCM can be found in the smoke of thermal degradation in minute quantities, with low risk.

Studies in the VCM and PVC show relative risks and odds ratios between 4 and 8 times, with high confidence interval. The IARC classifies vinyl chloride in Group 1, ie there is sufficient evidence on human carcinogenicity. Among the exposed in the same industry, also notes the presence of other work-related diseases, such as acro-osteolysis (Degeneration of the bones of the phalanges terminals), the Raynaud's syndrome, the scleroderma, the thrombocytopenia and changes in liver function.

The hepatic angiosarcoma should be classified as work related disease, the Group II of the Classification of Schilling, since the work can be considered as a risk factor in the set of risk factors associated with the etiology of this multifactorial tumor.

3. CLINICAL AND DIAGNOSTIC

The clinical picture is characterized by abdominal pain, palpable mass in the right upper quadrant, pain sensitivity in the right upper quadrant, weight loss and ascites. The highly vascular nature of hepatic angiosarcoma may cause massive hemorrhage peritoneal. Can be observed simultaneously, deterioration of liver function, obstructive jaundice with pruritus, mild cholecystitis, recurrent hepatitis or signs of metastatic disease. Most patients die due to liver failure or due to massive hemorrhage within the tumor.

The diagnosis of hepatic angiosarcoma is based on clinical history. In later stages, the physical examination can contribute.

CHAPTER 11

WORK-RELATED DISEASES PROCEDURES

MANUAL

FOR HEALTH SERVICES

In laboratory tests, the alphafetoprotein is elevated in 30 to 50% of cases, but is not pathognomonic, since this marker is also rising in other tumors. The liver function tests are generally altered, especially alkaline phosphatase (90% of cases). The AST and LDH are elevated in more than two thirds of cases, but the TGP is usually normal. Cirrhotic patients have a chronic elevation of transaminase levels, which may see a decrease when the tumor develops. Liver scintigraphy shows results difficult to interpret, but is useful in patients with tumors solitary early. Selective angiography of the hepatic artery allows for the differential diagnosis. Liver biopsy is the definitive diagnosis. In a solitary nodule, as evidenced in liver scintigraphy, we should perform an angiography before biopsy to rule out hemangioma or other highly vascularized lesions. Biopsies of vascular lesions should be performed by laparoscopy or laparotomy, to minimize the risk of bleeding. In the presence of multiple nodules on scintigraphy, biopsy can be performed percutaneously.

4. TREATMENT AND OTHER CONDUITS

The tumor malignancy is high. Surgical treatment (lobectomy), even in selected patients have poor prognosis. The tumor responds poorly to radiotherapy and chemotherapy. The median survival of patients with hepatic angiosarcoma is about five months. Many patients still die in hospital at the time of diagnosis. Patients who present tumors located survive longer.

5. PREVENTION

The prevention of angiosarcoma of the liver work-related based on the procedures for monitoring the environment and working conditions and the effects or damage to health, described in the introduction to this chapter. The elimination or control of exposure to vinyl chloride is fundamental to reducing the incidence of disease in groups occupational at risk.

Observe the principles of the Convention / ILO No. 139/1974:

- seek, in every way, replace the substances and carcinogens by other não cancerígenos or less harmful;

- reduce the number of employees exposed, duration and exposure levels to a minimum consistent with safety;
- prescribe measures;
- establish appropriate system of record;
- inform workers about the risks and measures to be applied;
- ensure that medical exams needed to evaluate the effects of exposure.

The environmental control measures aimed at eliminating or reducing exposure to levels close to zero or within the limits established by:

- entrapment and isolation procedures of work sectors;
- use of hermetically sealed systems in the industry;
- adoption of standards of hygiene and safety with stringent exhaust ventilation systems with appropriate and effective, systematic monitoring of the concentration in ambient air;
- changes in work organization that allow reducing the number of exposed workers and exposure time;
- measures of general cleaning of work environments, personal care, resources for bathing, washing hands, arms, face and exchange of clothing;
- provision by the employer of appropriate personal protective equipment in good repair, as indicated in a complementary way to measures of collective protection. The Occupational Safety and Health Administration (OSHA) sets the permissible exposure limit (PEL or LEP) for the vinyl chloride monomer 1 ppm (5 ppm for 15 minutes). The National Institute for Occupational Safety and Health (NIOSH), to include vinyl chloride between carcinogens and recommends that exposure be as low as possible. The exposure limit (TLV-TWA) for vinyl chloride, proposed by American Conference of Governmental Industrial Hygienists (ACGIH) is 1 ppm, with the notation of being a carcinogen classified as A1, or confirmed human carcinogen.

In Brazil, NR 15 still retains the LT of 156 ppm or 398 mg/m³ of vinyl chloride in ambient air, in apparent contradiction with the parameters currently recommended by OSHA, NIOSH and the ILO itself. It is urgent that this parameter is updated in both federal regulations and state or municipal regulations. It is recommended to check the suitability and adoption by the employer of the control measures of occupational risk factors and health promotion identified in the PPRA (NR 9) and PCMSO (NR 7), and other regulations - health and environmental - in the states and municipalities.

The periodic medical examination aims to identify signs and symptoms for early detection of disease. We recommend the use of standardized procedures and conducting tests of liver function, with dosage of serum transaminases (AST and ALT), lactate dehydrogenase (LDH), alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT) at baseline. Each year, the shutdown and the termination of worker exposure. Although this procedure does not reduce the incidence of hepatic angiosarcoma, may contribute to its detection in earlier stages, thus increasing the possibility of longer survival.

The cases detected should be reported. Due to the severity and rarity of the encounter case hepatic angiosarcoma in individuals exposed to vinyl chloride should be considered as a sentinel event.

Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the existing information systems in SUS, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

Malignant neoplasm of Pancreas ICD-10 C25. -

1. DEFINITION OF THE DISEASE - DESCRIPTION

The malignant neoplasms of the pancreas related to work cover the affections of the exocrine pancreas, mainly type carcinomas adenocarcinoma, localized in the pancreatic head in 60% of cases in the body, between 15 to 20% in the tail in 5% of cases, the diffuse or spread of high malignancy in 20%, and the endocrine pancreas, rarer.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The causes of Pancreatic cancer are unknown, but its occurrence in certain groups of people differently allows us to identify some risk factors, among which stands out smoking. It is estimated that this factor weighs about 50% in the etiology of pancreatic cancer. Alcoholism and pancreatitis are visibly associated with this malignant tumor and the recurrent hereditary pancreatitis. Diabetes mellitus seems to be a risk factor in some cases, but it is unclear whether this is true, since the Pancreatic cancer can also cause diabetes in older patients. Diets containing high levels of animal fat, especially meat, have been identified

as a risk factor for pancreatic cancer, as well as excessive coffee and vitamin A.

The etiology of occupational Pancreatic cancer has been investigated and the results are unclear so far. About 24 products or chemicals used or produced in workplaces, in about 14 industries and / or professions, have been linked to production and excess incidence and / or mortality such malignant tumor. However, few studies are methodologically consistent.

Among these include the studies on chemical workers exposed to DDT (dichloro-diphenyl-trichloroethane) among which was reported a relative risk of 5. Workers of the mechanical-metallurgical industry and automobile industry, exposed to mineral oils (soluble oils) also are more susceptible to pancreatic cancer in several well-conducted studies of methodological point of view. As occurs with many other locations, ionizing radiations in working environments are associated with pancreatic cancer in high-risk groups including radiologists.

The Pancreatic cancer can be classified as work related disease, the Group II of the Classification of Schilling and the work is considered as a risk factor in the set of risk factors associated with multifactorial etiology of this tumor.

3. CLINICAL AND DIAGNOSTIC

The cancer of the pancreatic tail can be asymptomatic for a relatively long time, since it does not affect the neighboring structures. The site of primary tumor often discovered only after evidence of distant metastasis. Nocturnal abdominal pain, which tends to get progressively worse, is the most common symptom of pancreatic cancer. Can be relieved with painkillers or, by assuming the antalgic position sitting with the torso leaning forward. May be associated with jaundice, weight loss, intestinal bleeding, commonly associated with tumors of pancreas and ampulla are rare in other tumors. Steatorrhea and diabetes mellitus, when they arise in the elderly, associated with progressive weight loss should alert to the possibility of pancreatic cancer. Hepatomegaly and abdominal mass appear late in the disease course.

The diagnostic investigation to assess the extent of disease and the presence of metastases who recommend surgery with curative purposes, includes hemogram, liver function tests, liver scintigraphy, bone scans, X-ray contrast (upper gastrointestinal series) upper gastrointestinal (seeking pyloric obstruction or deformity or duodenal ulcer) and biopsies of suspected metastatic masses.

Therapeutic results, once proven histopathologically the

pancreatic cancer are poor.

Workup is expensive, may increase morbidity and does not alter the prognosis for these tumor. Computed tomography of the abdomen detects 90% of cases of pancreatic cancer. Endoscopy is useful in carcinoma of the ampulla of Vater, a potentially curable lesion. Endoscopic cholangiopancreatography is an accurate method in

90 to 95% of cases, especially if the tumor is the head of the pancreas and if combined with other diagnostic studies. The cytological evaluation may improve sensitivity. Pancreatic function tests are no longer considered useful in diagnosis.

4. TREATMENT AND OTHER CONDUITS

Are indicated surgery, radiotherapy and chemotherapy, with discouraging results. The prognosis is always bad, being slightly affected by the location. Patients with cancer confined to the head of the pancreas have a relatively better prognosis than those who are located in other areas of the pancreas. As to histological grade, high-grade lesions to malignancy are associated with a survival of 3 months and the tumors of low histological grade of malignancy represent a survival of about 6 months. A median survival is 6 months, only in the presence of local extension, and two months in metastasis to other organs. The mortality rate exceeds 80% in the first year and not more than 1% of patients achieved 5-year survival.

5. PREVENTION

The prevention of malignant neoplasm of the pancreas related to work based on the procedures for monitoring the environment, working conditions and the effects or damage to health, described in the introduction to this chapter.

The control of occupational exposure to ionizing radiation, the DDT and mineral oil (oil soluble) mainly in mechanical and metallurgical industry, can reduce the incidence of disease in risk groups. The environmental control measures aimed at elimination of exposure and to control the levels of concentration of agents near zero, by: entrapment and isolation procedures of work sectors;

- hygiene standards and strict safety, local exhaust ventilation systems and general ventilation adequate, effective, systematic monitoring of concentrations of agents in ambient air;

- changes in work organization that allow reducing the number of exposed workers and exposure time;

- measures of general cleaning of work environments and facilities

for personal hygiene, resources for bathing, washing hands, arms, face, change of clothing, toilet cleaner and easily accessible;

- provision of appropriate personal protective equipment such as protective masks and other breathing, so additional measures for collective protection.

Mechanical and metallurgical industries and other processes with exposure to oil is recommended to provide the machinery and equipment shields to prevent splashing cutting oils reach the skin of workers.

Federal Law No. 7.802/1989 and some state and local laws prohibit the use of organochlorine pesticide, including among the insecticide DDT (dichloro-diphenyl-trichloroethane), must therefore not be allowed manufacturing and use. Exposure to ionizing radiation should be limited by strict control of radiation sources, both in industrial environments such as in health services. On specific procedures

For health surveillance of those exposed to ionizing radiation see in this chapter, the protocol malignant neoplasm of bone and articular cartilage of limbs.

It is recommended to check the suitability and adoption by the employer of the control measures of occupational risk factors and health promotion identified in the PPRA (NR 9) and PCMSO (NR 7), and other regulations - health and environmental - in the states and municipalities.

The periodic medical examination aimed at identifying signs and symptoms for early detection of cases.

In addition to careful clinical examination is recommended to use standardized instruments and laboratory testing, appropriate to the identified risk factor, including the CBC. Measures of health promotion and tobacco control should also be implemented.

Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the existing information systems of the SUS, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

Malignant neoplasm of the nasal cavity and paranasal sinuses ICD-10 and C30-C31. -

1. DEFINITION OF THE DISEASE - DESCRIPTION

Tumors affecting the nasal cavity and paranasal sinuses must be distinguished from tumors limited to the nasal cavity and those that arise from the paranasal sinuses. About 60% of these malignant tumors are found in the maxillary sinuses, nasal cavity in 20% and 15% in the ethmoid sinuses, nasal vestibule at 4% and 1% in the frontal sinus or sphenoid. Approximately 80% of malignant tumors this region develop in the mucosal surface and 54%, most are squamous cell carcinomas. They can also occur anaplastic carcinomas (17%), transitional cell carcinomas (7%), adenocarcinomas (6%), melanoma (5%), lymphoma (6%) and others.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Little is known about the etiology and predisposing factors, although it is considered that excessive consumption of alcohol, smoking and poor oral hygiene can play a facilitator role. The lag time is relatively long, often exceeding 20 or 25 years. Among the etiologic agents and risk factors of nature Occupational best known for these tumors are:

- ionizing radiation;
- chrome and compounds (salts probably hexavalent chromium);
- process of refining nickel;
- production isopropyl alcohol (Strong acid process, probably by exposure to alkyl sulfate produced in the process);
- dust from wood and other organic dusts the furniture industry (production of adeno-carcinomas);
- dust of the leather industry;
- organic dusts (in textile industry and in bakeries)

The malignant tumors of nasal and / or paranasal sinuses can be classified as work-related diseases, the Group II of the Classification of Schilling and the work is considered as a risk factor in the etiology of these multifactorial malignant tumors.

3. CLINICAL AND DIAGNOSTIC

Signs and symptoms resemble those of sinusitis, including local pain, soreness, toothache, bloody nasal discharge, tooth decay, bad adjustments of dentures. May arise, though, visual disturbances, proptosis, nasal obstruction, protruding mass on the cheek, which may ulcerate through the skin and palate.

Rhinoscopy, sinoscopy and CT or MRI of the affected area

should be accomplished. The presence of bone destruction on X-ray, fosters the suspicion of a neoplastic process, although it can occur in certain benign conditions.

4. TREATMENT AND OTHER CONDUITS

Surgery is indicated because of the frequency of bone involvement. Radiotherapy is almost always needed because the margins left by resection is usually narrow. In tumors of the nasal cavity, preferably with radiotherapy for the treatment of early lesions.

If there is bone involvement, or if the tumor is a melanoma or sarcoma, surgery is indicated. In advanced lesions, indicates the combined surgical resection and radiotherapy. Follow-up studies of patients who underwent surgery, radiotherapy combined treatment showed, on average, a 5-year survival by about 40% of patients. Well located and defined cases may reach a longer survival.

The criteria for assessing and internship disability produced by disease, when present, includes: olfaction disorders, respiratory disorders, nasal stenosis, mutilating injuries and loss of substance, rhinorrhea or combined forms. The development of parosmia (Off-flavors) or anosmia residual post-treatment may cause significant impacts on workers, both in their defense mechanisms, in exposures to toxic chemicals or dangerous, as in their ability to work, depending on their job. Any cosmetic damage may be recovered in view of the Medical Insurance and the civil and criminal legislation.

5. PREVENTION

The prevention of malignant neoplasm of the nasal cavity and paranasal sinuses related to work based on the procedures for monitoring the environment, working conditions and the effects or damage to health, described in the introduction to this chapter.

Environmental control of exposure to chrome andnickel, nickel and dust from wood, and other agents, can effectively reduce the incidence of disease in occupational groups at risk. The environmental control measures aimed at eliminating or reducing the exposure concentration levels approaching zero, through:

- entrapment and isolation procedures of work sectors, or the use of hermetically sealed systems;
- hygiene standards and strict safety and cleanliness of workplaces, with the wet cleaning or water cleaning of environmental surfaces (countertops, walls, floors) or by suction, to remove particulates before the commencement of activities;

- exhaust ventilation systems adequate, effective and systematic monitoring of concentrations of smoke, haze and dust in the air;

- changes in work organization that allow reducing the number of exposed workers and exposure time;

- in mining activities, and these must be taken to wet drilling techniques to reduce dust concentrations in ambient air and the use of respiratory protective masks. If levels are above acceptable, may be necessary to use air purifying equipment;

- provision by the employer of personal protective equipment in order to supplement the measures of collective protection. The respiratory protective masks should be used as a measure temporary emergencies. When the measures of collective protection is inadequate, they should be carefully indicated for some sectors or functions. Workers must be trained appropriately for their use. Masks should be of quality and relevance to exposures with chemical or dust filters, specific for each substance handled or groups of substances which may be retained by the same filter. Filters should be changed strictly according to the manufacturer's recommendations. The Normative / MTb No. 1 / 1994 lays down rules on the use of technical equipment for respiratory protection.

For specific procedures for health surveillance of those exposed to ionizing radiation see in this chapter, the protocol Malignant neoplasm of bone and articular cartilage of limbs.

It is recommended to check the adequacy of and compliance by the employer, the PPRA (NR 9), the PCMSO (NR 7) and other regulations - health and environmental - in the states and municipalities. It is recommended to query the NR 15, which defines the LT concentrations in ambient air of various chemicals, for journeys of 48 hours a week. Annex No. 11 NR 15 provides for the LT nickel carbonyl 0, 04 ppm or 0, 28 mg/m³ of air.

The periodic medical examination aimed at identifying signs and symptoms for early detection of disease. In addition to thorough clinical examination, we recommend the use of standardized instruments and laboratory testing appropriate to the risk factor identified. Although the concentration of hexavalent chromium urine not keeping with the risk of cancer, the IBMP is expected to be 30g / gcreatinine urine, and VR for non-occupationally exposed populations is up to 5 g / gcreatinine. Although the holding of periodic medical examinations do not reduce the incidence of cancer of nasal and paranasal sinus related (or not) to work, can contribute to its detection at earlier stages, thus increasing the success of treatment.

Suspected or confirmed the relationship of disease with the work,

you must:

- inform the employee;
- examine exposed in order to IDENTIFYING other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;
- provide for the issuance of the CAT, if the employee is insured SAT by Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

Malignant neoplasm of bronchus and lung C34 ICD-10. -

1. DEFINITION OF THE DISEASE - DESCRIPTION

The group of diseases encompassed in lung cancer comprises:

- squamous cell carcinoma, also known as squamous cell carcinoma, accounts for about 30% of all malignant neoplasm of the lung, most commonly central (80%) than peripheral (20%);

- small cell carcinoma, accounts for 20% of malignant lung cancer, the most frequent location mediastinal or hilar (95%) than peripheral (5%);

- adenocarcinoma and large cell carcinoma, accounts for about 30% of all malignant lung cancer, the most frequent location in the periphery, such as peripheral nodules (70%);

- histologically mixed cancer, accounting for about 20% of all malignant lung cancer;

- lung tumors uncommon (bronchial carcinoids, adenoid carcinomas, cisticose carcinosarcomas).

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

From the studies on the etiology of lung cancer made by Doll & Hill in 1950, numerous studies have shown that smoking is the most important cause of neoplasm, accounting for approximately 80-90% of cases. Smokers have a risk of dying from lung cancer increased by about 10 times, on average, whereas in heavy smokers, the relative risk is 15 to 25 times. The carcinogens more

known, produced by combustion of tobacco, are tobacco-specific nitrosamine and polycyclic aromatic hydrocarbons. Other risk factors are documented in the literature: industrial pollution, living in densely urbanized and non-occupational exposure to ionizing radiation. The risk of lung cancer attributable to occupation ranging from 40 to 40%, according to the agent examined. However, there remain issues to be

better explained, especially with regard to the interference of smoking as a confounding variable and the nature of the combination of effects, additive or multiplicative.

The etiologic agents and risk factors of occupational nature best known are:

- arsenic arsenic and its compounds;
- asbestos or asbestos. Should be investigated current and past exposure, considering even small exposures over the years, for example, plumbers who install water tanksasbestos cement, making the holes for the passage of pipes and breathing in dust; carpenters construction, by fixing the tilesAsbestos cement with screws, mechanical sanding pads and brake pads; exposure to talc contaminated with fibers asbestos industrial rubber products, sanding the putty used to repair various objects (a plastic mass may contain talc contaminated by asbestos in its composition), among many others;

- beryllium;
- cadmium or its compounds;
- chrome and its toxic compounds;
- vinyl chloride. It is present in plantsvinyl chloride in the production of PVC (polymer) or

Exposure to vinyl chloride monomer (VCM);

- chloromethyl ethers;
- free silica;
- tar, pitch, bitumen, mineral coal, paraffin and waste products such substances;

- ionizing radiation;
- emissions from coke ovens (polycyclic aromatic hydrocarbons);
- nickel compounds. Threatening insoluble compounds and complexes nickel with carbon monoxide. The operation of stainless steel welding fumes can lead to high levels ofnickel;

- acrylonitrile. In the form of monomer used in the chemical industry;

- formaldehyde. Theformaldehyde (Formaldehyde or formaldehyde) is volatile and very used to preserve tissue

in anatomy laboratories, as raw material in some processes in chemical industry, or are derived from the polymerization reaction of some synthetic resins, for example, Sintek

- Processing (casting) of Aluminum and other metals;

- mists from mineral oil (cutting oil or oil soluble).

Since 1955 it recognized the causal relationship between exposure

to asbestos or asbestos and the occurrence of mesothelioma of the pleura, peritoneum and lung cancer, associated or not with asbestosis. Occupational exposure to asbestos - The most important in producing work-related lung cancer - Produces an increase of 3 to 4 times the risk of lung adenocarcinoma workers in non-smokers and carcinoma

Squamous cell workers in smokers (risk 3 times the risk of non-smokers exposed to asbestos). Thus, workers exposed to smokers/asbestos, the relative risk (synergistically multiplied) is increased

in 90 times. Strict epidemiological studies have shown, from the 50s, the importance of hexavalent chromium, or chromium ion $6 +$ valence CrVI or, in the etiology of lung cancer. The exhibition takes place, particularly in the production of chromium, in the mists of the plating tanks, paint pigments, aschromates of lead and zinc, welding fumes from metals with high content of chrome, stainless steel and electroplating processes and industries ferro-chrome.

Ionizing radiations are historically associated with malignant tumors. His contribution in the etiology of lung cancer has been described in healthcare workers (radiologists) for underground mines iron, with exposure to radioactive radon, inestin, uranium, probably gold and workers

mines coal. The lag time is relatively long, rarely less than 15/20 years.

The lung cancer can be classified as work related disease, the Group II of the Classification of Schilling and the work is considered a risk factor associated with the etiology of multifactorial lung cancer.

3. CLINICAL AND DIAGNOSTIC

A history suggestive of lung cancer includes smoking, onset of cough or change in the pattern of previously existing cough, hoarseness, hemoptysis, anorexia, weight loss, dyspnea, pneumonia resolution dragged, chest pain and symptoms of paraneoplastic syndromes. The apical location and presence of metastases

They can produce clinical polymorphs.

The diagnosis of lung cancer is based on clinical history, physical examination and laboratory tests, particularly chest X-rays, computed tomography (CT) scan, sputum cytology and endoscopic procedures with sample collection and examination histology, because the approach adopted will depend on the histologic type of tumor, as well as its staging.

4. TREATMENT AND OTHER CONDUITS

The recommended treatments are:

- lung resection surgery for partial or total;
- radiotherapy;
- chemotherapy.

The correct diagnosis of lung cancer allows staging of the tumor in relation to prognosis and survival, from the extent of disease, performance status of the patient, Status Performance and tumor histology.

5. PREVENTION

The prevention of malignant neoplasm of bronchus and lung-related work has reference to the Convention / ILO No. 139/1974, which determines the adoption of the following measures:

- seek to replace all forms substances and carcinogens by other não cancerígenos or less harmful;
- reduce the number of employees exposed, duration and exposure levels to a minimum consistent to security;
- prescribe measures;
- establish appropriate system of record;
- inform workers about the risks and measures to be applied;
- ensure that medical exams needed to evaluate the effects of exposure.

Environmental control of arsenic, beryllium, chromium, nickel, cadmium, vinyl chloride, acrylonitrile, chloromethyl

ethers, formaldehyde, among other chemicals, can effectively reduce the incidence of disease in occupational groups at risk. The environmental control measures aimed at elimination of exposure and control of concentration levels approaching zero, through:

- entrapment and isolation procedures of work sectors;
- use in industry, hermetically sealed systems;
- hygiene standards and strict safety measures and general cleaning of work environments with wet cleaning or washing with water surfaces (countertops, walls, floors) or by suction, to remove particles prior to the commencement of activities;
- exhaust ventilation systems adequate and efficient;
- systematic monitoring of concentrations of smoke, haze and dust in the air;
- in mining, adopt wet drilling techniques to reduce dust concentrations in ambient air;
- changes in work organization that allow reducing the number of

exposed workers and exposure time;

- personal hygiene facilities, resources for bathing, washing hands, arms, face and exchange of clothing;

- provision by the employer of appropriate personal protective equipment in good repair in a complementary way to collective protection measures adopted.

The respiratory protective masks should be used as a temporary measure in emergencies. When the measures of collective protection is inadequate, these should be carefully indicated for some sectors or functions. Workers must be trained appropriately for their use. Masks should be of quality and relevance to exposures with chemical or dust filters, specific for each substance handled or groups of substances which may be retained by the same filter. Filters should be changed strictly according to the manufacturer's recommendations. The Normative / MTb No. 1 / 1994 lays down rules on the use of technical equipment for respiratory protection.

It is recommended to check the suitability and adoption by the employer of the control measures of occupational risk factors and health promotion identified in the PPRA (NR 9) and PCMSO (NR 7), and other regulations - health and environmental - in the states and municipalities.

Annex 11 NR 15 (Ordinance / MTb No. 12/1983) sets out the LT for some chemicals in the air, for journeys of up to 48 hours per week. Among the agents known to cause malignant neoplasm of bronchus and lung-related work are:

- Arsine: 0, 04 or 0 ppm, 16 mg/m³;
- vinyl chloride: 156 ppm or 398 mg/m³;
- formaldehyde: 1, 2 or 6 ppm, 3 mg/m³;
- nickel carbonyl: 0, 04 ppm or 0, 28 mg/m³.

These limits should be compared with those adopted by other countries and reviewed periodically in light of current knowledge and evidence. It has been observed that even when strictly followed, do not prevent

the appearance of damage to health.

The periodic medical examination aimed at identifying signs and symptoms for early detection of disease in addition to clinical examination, we recommend the use of standardized instruments such as questionnaires on respiratory symptoms already validated, and appropriate laboratory tests. Measures of health promotion and tobacco control should also be implemented.

Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notifying appropriate information systems in health (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;
- arrange the issue of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

Malignant neoplasm of bone and cartilage ARTICULAR

MEMBERS (Including Bone Sarcoma) ICD-10 C40 .-

1. DEFINITION OF THE DISEASE - DESCRIPTION

Sarcomas are malignancies of mesenchymal tissues. Osteosarcoma or osteogenic sarcoma is a primary malignancy of bone, consisting of malignant stromal osteoblasts forming osteoid. The Classic Osteosarcoma a tumor is poorly differentiated, highly aggressive, affecting mostly young adults, involving most frequently the long bones (femur, tibia and humerus) and can be classified as osteoblastic, chondroblastic or fibroblastic, according to the predominant histologic component

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The causes of osteosarcoma are not known. In Classic osteosarcoma primary the young person under 20 years of age, development occurs in apparently no other bone pathology, arising from the metaphyses of long bones, before closure of the epiphysis.

The Secondary osteosarcoma develops in older people both in flat bones and long bones, usually superimposed on a pre-existing bone pathology, such as: disease Paget, enchondromas, exostosis, osteomyelitis, fibrous dysplasia, heart attacks and fractures, or a result of exposure to carcinogenic agents

environment, especially ionizing radiation, exposure to environmental, occupational or iatrogenic. Patients with familial retinoblastoma are at increased risk of developing osteosarcoma.

It's classic history of occupational exposure to ionizing radiation workers in factories and workshops of clocks and similar instruments, with figures, signs, dials and luminous hands and luminescent. The ink used contained radium on zinc sulfide and wet work and adjusted the little brush in the mouth, enrolling as a result, numerous cases of radionecrosis of the mandible, aplastic anemia and osteosarcoma. Whereas this process

work was abandoned, the incidence of osteosarcoma related to working with these characteristics tend to disappear.

The occurrence of osteosarcoma in adult workers with a history of occupational exposure to ionizing radiation should be classified as work related disease, the Group I of the Classification of Schilling and the work is deemed necessary cause in the etiology of these tumors, although other risk factors may act as adjuvants.

3. CLINICAL AND DIAGNOSTIC

The clinical picture is characterized by local pain, inflammation and swelling in the region corresponding to the tumor. In primary osteosarcoma the young man, struck by the persistent pain component, more at night, before outward signs. The diagnosis is based on history, local examination, radiography, computed tomography and measurement of alkaline phosphatase. Should be investigated lung metastases.

4. TREATMENT AND OTHER CONDUITS

The specialized treatment is surgical, combined radiotherapy and chemotherapy.

5. PREVENTION

The prevention of malignant neoplasm of bone and articular cartilage of the members of work-related should be guided by the principles of the Convention / ILO No. 139/1974, concerning the prevention and control of occupational hazards caused by substances or agents carcinogens, ratified by Brazil in June 1990 and in force since June 1991, mentioned in the introduction to this chapter. Environmental control of exposure to ionizing radiation is essential to reduce the incidence of disease in occupational groups at risk. Exposure to ionizing radiation should be limited, with strict control of radiation sources, both in industrial environments such as in health services. Observe the following basic guidelines for radiological protection in medical and dental radiology, as defined by Ordinance / MS No. 453/1998:

- the equipment should have safety devices, protective shields and rigorous preventive maintenance;
- rooms and areas shall be provided with signage, and screen;
- operating procedures and safety must be well defined, including accident and emergency situations;
- personnel must receive proper training and be supervised;
- equipment and supplies must be positioned as far as possible from workers;

- must reduce the number of workers in these sectors and the exposure time.

Workers exposed to ionizing radiation should be ensured:

- continuous monitoring by means of patient dose;
- periodic examinations to detect early health effects, including complete blood count and platelet count in the pre-employment and every six months;

- provision of protective equipment, including, shields, aprons and gloves shielded. Monitoring procedures should include the adequacy of and compliance by the employer, the PPRA (NR 9), the PCMSO (NR 7) and other regulations - health and environmental - in the states and municipalities. The holding of periodic blood counts in workers exposed to ionizing radiation, as determined

NR 7, although not help reduce the incidence of osteosarcoma and other malignancies, may contribute to early detection of other signals related to overexposure.

Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;

- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;

- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

OTHER MALIGNANT NEOPLASM OF THE SKIN ICD-10 C44. -

1. DEFINITION OF THE DISEASE - DESCRIPTION

The epitheliomas are epithelial neoplasms, which may be benign or malignant. However, the term epithelioma is usually reserved to describe malignant processes, corresponding to basal cell carcinomas (Basal) and squamous cell carcinomas (Squamous). The melanomas are usually included in another category.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The etiology of skin cancers is strongly associated with actinic exposure, especially ultraviolet rays. About 90% of these cancers develop in regions of the body exposed to the sun. The incidence in white

populations is greatly increased in regions near the equator and with altitude compared with sea level. The fair-skinned people, who suffer sunburn more easily, have an increased risk of developing skin cancer. The incidence in blacks is much lower than in whites. Occupations that expose workers to intense solar radiation, as farmers, construction workers and open pit mining, fishermen and sailors, for example, have rates of skin cancer higher than the general population or workers in other occupations that are less exposed to actinic radiation. Other risk factors predisposing or have been observed, such as family history of skin cancer, receiving a transplanted kidney, xeroderma pigmentosum, Gorling syndrome, albinism, infections with human papillomavirus (HPV), chronic inflammation, scarring, arsenic keratoses (Bowen's disease,) Solar keratoses and trauma.

The skin cancer due to occupational exposure was described for the first time, by Percival Pott in 1775, workers in the scrotum cleaners (or ex-cleaners) chimneys, after direct skin contact with soot. Later in 1915, Yamagiwa & Ichikawa described the induction of skin tumors in animals by applying coal tar on their skin. In the 40s, was isolated and synthesized the benzopyrene (3, 4 - benzopyrene) identified as responsible for cancerous tumors described by Pott. Today, it is known that different polycyclic aromatic vary widely in their carcinogenic potency. Another example is the skin cancer due to arsenic, either in their production, use of its products or drinking contaminated water, as occurs endemically in northern Argentina, Chile, Mexico and Taiwan regions. Ionizing radiation can also cause skin cancer, even with current security procedures is believed that the incidence has dropped markedly.

The following etiologic agents and risk factors of occupational nature should be considered in the investigation of the etiology of skin cancer workers:

- arsenic and its compounds;
- tar, pitch, bitumen, mineral coal, paraffin, creosote, tar sands, oil shale and waste products such substances;
- ionizing radiation;
- ultraviolet radiation;
- mineral oils, lubricants and cutting fluids or paraffin.

The malignant epitheliomas can be classified as work-related diseases, the Group II of the Classification of Schilling and the work is considered an important risk factor associated with multifactorial etiology.

3. CLINICAL AND DIAGNOSTIC

The basal cell carcinoma, or basal cell, is presented as a rough injury pigmented telangiectasia, slow growing and localized at sites of exposure. The Squamous cell carcinoma or squamous manifests itself as a nevus erythematosus, slow growth, which may progress to nodules that frequently ulcerate. The manifestations of the distant carcinomas are rare and can result in loss of weight,

anorexia, lethargy, pleural effusion, ascites, neurological symptoms caused by metastases and bone pain.

Diagnosis is by history and physical examination with emphasis on skin, in the back, oral cavity, perianal and genital lesions, intertrigo, search adenopathy in the neck auscultation, abdominal palpation for detection of tumor masses and hepatomegaly. Laboratory tests including hematological

complete hepatic transaminases, alkaline phosphatase and biopsy injury.

The best diagnostic feature of basal cell carcinoma is biopsy of the suspicious lesion. When there is suspicion of squamous cell carcinoma, biopsy should be deepened.

The differential diagnosis should be done with some infections (fungal infections, tularemia, syphilis, anthrax) inflammatory lesions (pyoderma gangrenosum, drop), venous stasis and varicose ulcers, psoriasis, seborrheic keratosis and premalignant.

4. TREATMENT AND OTHER CONDUITS

It is indicated to traditional surgical resection, with a margin free of tumor 3 to 10 mm, depending on their size. Other procedures include Mohg surgery, curettage, cryosurgery and radiotherapy. We recommended in large tumors and deep erosion, or with distant metastasis, should be evaluated medically and surgically with due discretion. In general, the success of surgery and radiotherapy of epithelioma malignant, non-metastatic, is extremely high, reaching ranges between 90 and 95%. There may be recurrence of the tumor, especially if the excision was not done with proper safety margin.

In skin tumors, as in other skin diseases, the deficiency, if any, may relate to the functional sphere, itself, and the aesthetic sphere. In the first, depending on the degree of the lesion and its location, there may be loss of movement and other functions related to daily activities. Pain and itching may be important. After surgical treatment, may remain sequelae of disfigurement of the patient, signs of scars and skin grafts.

5. PREVENTION

The prevention of malignancy work-related skin based on the procedures for monitoring the environment, working conditions and the effects or damage to health, described in the introduction to this chapter.

The elimination or control of exposure to arsenic, the derivatives mineral coal and the coke oil, contact with mineral oils and derivatives exemplify and radiological protection measures that can reduce the incidence of malignant epitheliomas occupational groups at risk. Control measures aimed at eliminating environmental exposure or reduction to concentrations close to zero, by:

- entrapment and isolation procedures of work sectors;
- use in industry, hermetically sealed systems;
- hygiene standards and strict safety;
- general cleaning arrangements of the work environment, facilities for personal hygiene, as resources for bathing, washing hands, arms, face and exchange of clothing;
- exhaust ventilation systems adequate and efficient;
- systematic monitoring of concentrations of smoke, haze and dust in the air and ionizing and non-ionizing;
- Wet drilling techniques in mining activities to reduce dust concentrations in ambient air;
- changes in work organization that allow reducing the number of exposed workers and exposure time;
- provision by the employer of appropriate personal protective equipment in good repair, so additional measures for collective protection.

Among the specific preventive measures to control exposure to ultraviolet radiation, are:

- gradual exposure to solar radiation;
- limiting exposure to times of lower solar radiation;
- use of sunscreen (sunscreens) that absorb ultraviolet (UVB);
- proper clothing, clean, airy fabric suitable climatic conditions (temperature and (Humidity), including hats, to protect the face and body skin exposure outdoors.

On procedures for monitoring of exposure to ionizing radiation see in this chapter, the protocol Malignant neoplasm of bone and articular cartilage of limbs.

It is recommended to check the suitability and compliance by the employer, the PPRA (NR 9), the PCMSO (NR 7) and other regulations - health and environmental - in the states and municipalities. The periodic

medical examination aimed at identifying signs and symptoms for early detection of disease. It consists of clinical evaluation and complementary tests guided by occupational exposure. For some agents, NR 7 sets specific parameters, for example, for the IBMP arsenic urine is 50 g / gcreatinine.

The holding of periodic medical examinations, with strict dermatological examination among workers at risk groups, but not reduce the incidence of skin carcinomas related (or not) to work can contribute to its detection in early stages, increasing the treatment success.

Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

Mesothelioma: ICD-10 C45. -

Mesothelioma of the pleura C45. 0

MESOTHELIOMA OF PERITONEUM C45. 1

MESOTHELIOMA OF PERICARDIUM C45. 2

1. DEFINITION OF THE DISEASE - DESCRIPTION

Mesotheliomas are tumors - benign or malignant - of mesodermal origin, arising in the lining layer of the pleural cavities, pericardial or peritoneal.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The etiologic relationship of malignant mesothelioma with asbestos (Asbestos) was established by Wagner and colleagues at work in the Cape Province, South Africa, published in 1960. Later, New house and employees, the UK, confirmed this causal relationship, in a classic epidemiological study, cases of type x controls, held in London in the 70s. Both studies showed that both the occupational exposure toasbestos and environmental exposure in homes near industrial plants and / or exposure of women workers through the contaminated clothing fiber asbestos

brought from the factories, are associated with the etiology of malignant mesothelioma.

It is very high, over 90%, the probability that adults who develop Malignant mesothelioma of the pleura or peritoneum have worked or resided exposed to asbestos. The exposure should be investigated in the past of the patient, including small exposuresOver the years, such as plumbers who install water tanks asbestos cement, making the holes for the passage of pipes and breathing in dust; carpenters construction, drilling tilesAsbestos cement to mount, vehicle mechanical sanding pads and brake pads; workers exposed to talc contaminated with fibers asbestos activities in the industry of rubber and sanding putty used in the repair of numerous objects.

All types of fiber asbestos are carcinogenic to the production of malignant mesothelioma, being considered as complete carcinogens, since they act as initiators and as promoters of the process. Among the fibers, theamphibole (Crocidolite, anthophyllite, tremoliteandamosite, mostly) are the most responsible for production mesotheliomas.

The development of these malignancies does not appear to be dose dependent, which means that in principle, any number of fibers can initiate and promote tumor, which would explain its impact on women workers, their children or persons residing or frequent-covered buildings Asbestos, used for thermal insulation.

The latency period between initial exposure and manifestation of Malignant mesothelioma is very long and may be 35 to 45 years, although some studies show relatively short periods, around 20 years, but rarely less than 15 years.

The malignant mesothelioma of the pleura, peritoneum and / or pericardium, occurring in workers from occupational exposure toasbestos, should be classified as work-related diseases, the Group I of the Classification of Schilling, in which work can be regarded as a necessary cause in the etiology of these tumors, although other risk factors may act as coadjuvantes.Ter resided near plants that processasbestos may constitute an additional risk factor.

3 CLINICAL AND DIAGNOSTIC

Thepleural malignant mesothelioma appears as a small area in the form of plaque or nodule in the parietal or visceral pleura, which ultimately shaped coalescing, forming bulky tumor masses more frequently accompanied by pleural effusion.

The tumor develops by direct extension, forming large masses of

tumor invading adjacent structures, including the chest wall, the interlobar fissure, the pulmonary parenchyma, the mediastinum, pericardium, diaphragm, esophagus, great vessels of the mediastinum, contralateral pleura, and peritoneal cavity. Death is usually caused by compression of one or more of the vital structures.

In malignant mesothelioma peritoneal Thickening of the visceral and parietal peritoneum can surround and compress the intestine, liver and spleen. Large masses can cause intestinal obstruction and, in large expansions, the tumor extends into the retroperitoneum, the pancreas compressing the kidney and may invade the diaphragm and reach the lungs.

From the standpoint of cancer, mesothelioma can be classified as epithelial in about 35 to 40% of cases; sarcomatoid about 20% of cases; mixed in about 35 to 40% of cases and undifferentiated in about 50-10% of cases.

The clinical picture of malignant pleural mesothelioma manifested by dyspnea, chest pain or a combination of both symptoms. In the case of Malignant mesothelioma of the pericardium, the table may be chest pain and congestive heart failure, with findings of cardiac constriction, with increased cardiac shadow due to stroke, similar to pericarditis. The Malignant mesothelioma of the peritoneum it presents a framework of progressive ascites, abdominal pain and presence of tumor in the abdomen.

4 TREATMENT AND OTHER CONDUITS

The recommended treatments are:

- surgery;
- radiotherapy;
- chemotherapy.

The results are poor and median survival is 3 to 6 months regardless of treatment attempts.

5 PREVENTION

The prevention of work-related mesothelioma must follow the provisions of the Convention / ILO No. 139/1974, mentioned in the introduction of environmental controle asbestos (Asbestos) and, hopefully, the phase-out of its extraction, import, processing and use (as occurs with amphibole in Brazil and also with the chrysotile in many countries) may effectively reduce the incidence of Mesothelioma in exposed workers. The environmental control measures aimed at the elimination of or exposure to their control in near-zero levels through:

- enclosure of processes and isolation of sectors work, use of negative pressure and air humidification;
- use in industry, hermetically sealed systems;
- hygiene standards and strict safety measures for general cleaning of work environments and facilities for personal hygiene;
- exhaust ventilation systems adequate and efficient, with systematic monitoring of the fiber concentrations in ambient air;
- in mining, adopt wet drilling techniques to reduce dust concentrations in ambient air;
- changes in work organization that allow reducing the number of exposed workers and exposure time;
- provision by the employer of appropriate personal protective equipment in good repair in a complementary way to collective protection measures adopted. The use of masks or bullets to breathe (lung water) can be useful in jobs where there is intermittent exposure and brief.

The respiratory protective masks should be used as a temporary measure in emergencies.

When the measures of collective protection is inadequate, these should be carefully indicated for some sectors or functions. Workers must be trained appropriately for their use. Masks should be of quality and relevance to exposures with chemical or dust filters, specific for each substance

manipulated or groups of substances which may be retained by the same filter. Filters should be changed strictly according to the manufacturer's recommendations. The Normative / MTb No. 1 / 1994 lays down rules on the use of technical equipment for respiratory protection. OSHA sets the permissible exposure limit for all fibers asbestos greater than 5

microns, the value of 0, 1 fiber / cc, even exposure limit recommended by NIOSH. The ACGIH established in 1998 as an acceptable exposure limit (TLV-TWA) for all forms of asbestos, the same value (0, 1 fiber / cm³) with the observation that theasbestos should be considered confirmed human carcinogen (group A1).

In Brazil, NR 15 states since 1991, prohibiting the use of fiber amphibole (Crocidolite, amosite, tremolite), but for respirable fiberschrysotile LT sets of 2, 0 fiber / cm³. Federal Law No. 9.055/1995 discipline extraction, processing, utilization, marketing and transportation of asbestos / asbestos and products containing it, as well as natural and artificial fibers of any origin, used for the same purpose.

Prohibits extraction, processing, utilization and marketing of varieties belonging to the group amphiboles, the spraying of all types of

fibers and the sale of bulk fiber powder. Also specifies that all companies that handle or use materials containing asbestos / asbestos variety chrysotile or natural and artificial fibers shall submit annually, SUS listing of its employees, with indication of sector, function, position, date of birth, admission and periodic medical assessment and diagnosis. Indicates that the LT should be reviewed annually, maintained the lowest feasible and that transportation must comply with the transport of dangerous goods. The sectors of surveillance SUS will charge companies in their territory comply with the provisions of that law and taking measures to prevent the aggravation. In addition to thorough clinical examination is recommended:

- using standardized instruments such as questionnaires on respiratory symptoms previously validated national or internationally;
- Chest X-rays, the standard ILO (1980) at baseline and annually;
- spirometry, biennially, using the technique advocated by American Thoracic Society (1987).

It is important to reaffirm that these tests can be used for early diagnosis of asbestosis, does nothing for the pleural mesothelioma, always quick considering its emergence and evolution of highly lethal.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

Is underway in the country, following an international movement, a process of banning the use ASBESTOS.

MALIGNANT NEOPLASMS OF THE BLADDER ICD-10 C67

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1. DEFINITION OF THE DISEASE - DESCRIPTION

Bladder cancer encompasses a wide spectrum of neoplastic diseases, which includes tumors curable with minimal intervention, to invasive and metastatic those that lead to death. This spectrum of possibilities is representative of the likely evolution of a normal epithelium to a atypical epithelium

- a) carcinoma in situ
transitional cell carcinoma (Grade I and II)
- b) transitional cell carcinoma (Grade III)
- c) carcinoma paradoxicum.

From the histological point of view, about 90% of bladder cancers are Transitional cell carcinomas (Urothelial) and about 8% are subtypes of squamous cell carcinomas. Adenocarcinomas, sarcomas,

lymphomas and carcinoid tumors are very raros. Do clinical point of view and cystoscopic, the types of bladder cancer include papillary cancers solitary the most common and least likely to show infiltration, the diffuse papillary carcinomas, the sessile tumors and the carcinoma in situ.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Among the risk factors related to the production of these tumors include smoking, which increases from 2 to 3 times the risk of developing bladder cancer, coffee consumption, not yet fully proven, multiple urinary tract infections; bladder infestation by *Schistosoma hematobium*; abuse of phenacetin consumption and the use of cytotoxic drugs, as cyclophosphamide and chlorpromazine.

The etiology of chemical bladder cancer was proposed in 1895 by Rehn, Germany, when he noted that workers who produced dyes anilines had an increased incidence of tumor. Four decades later, HuEPO and colleagues induced bladder cancer in dogs exposed to beta-naphthylamine pure. Later, the observations reached benzidine and 4-aminobiphenyl, workers in rubber industry. Case and colleagues in the UK, established in 1954, through classic epidemiological study of historical cohort, the final confirmation of the causal association between bladder cancer and occupational exposure to various aromatic amines, hitherto been widely used as colorants or dyes.

Exposure to Polycyclic aromatic hydrocarbons, arising from smoking or occupational, has also associated the etiology of bladder cancer in some risk groups, such as workers exposed to coke oven emissions.

The following etiologic agents and risk factors of occupational nature should be considered in the investigation of the etiology of bladder cancer workers:

- tar, pitch, bitumen, mineral coal, paraffin and waste products such substances;
- aromatic amines and its derivatives (Beta-naphthylamine and 2-chloroaniline, benzidine, o-toluidine, 4-chloro ortho-toluidine) ;

- emissions from coke ovens;
- mineral oils or cutting soluble.

The Malignant tumors of the bladder can be classified as work-related diseases, the Group II of the Classification of Schilling and the work is considered a risk factor associated multifactorial in etiology.

3 CLINICAL AND DIAGNOSTIC

Haematuria is the main signal bladder cancer. About 50% of patients with gross hematuria and 1, 8 to 11% of those with microscopic haematuria have bladder cancer. There may also, obstruction of urinary frequency and bladder trigone, which impaired the elimination of urine. Specialized tests include

cystoscopy followed by biopsy, excretory urography, urine cytology and liver scintigraphy, splenic and bone, they search for metastases.

The diagnosis of bladder cancer is made from clinical history, physical examination and general urology, including digital rectal examination.

The differential diagnosis must be made with many other diseases that can cause hematuria and irritative bladder symptoms such as urinary infections, urolithiasis, benign prostatic hypertrophy, trauma and other cancers of the urinary tract

4 TREATMENT AND OTHER CONDUITS

Are indicated surgery, radiotherapy and chemotherapy. A malignant tumor is high, with high mortality in 3 to 6 months.

The diagnosis of bladder cancer, by itself, is insufficient to give an idea of the severity of the impact on the patient's performance and even prognosis in terms of *sobrevida*. *Tampouco* is associated with the inability of the patient lead a normal life, or almost normal, the overall performance of the life and professional activities. Systems can be used for staging, in the case of bladder cancer, most are based in *Chad* and less on clinical information histopathologic sections obtained at surgery. Regarding evolution, it is observed that:

- untreated patients have a 2-year survival of less than 15%, and median survival is 16 months;
- the squamous carcinomas and adenocarcinomas had worse prognosis than those transitional cell carcinomas;
- muscle invasion, lymph vessels and / or perivesical fat is associated with a worse prognosis. The invasive cancer is associated with a mortality rate of 50% in the first 18 months after diagnosis;

- carcinoma in situ progresses to invasive cancer in 80% of patients in the 10 years following diagnosis;
- histological grade of the tumor alone affects the survival of patients with superficial tumors. The 5-year survival is 85% in low grade lesions and 30% in high-grade lesions of malignancy. Virtually all the tumors of high malignancy, even surface, they become invasive, if untreated.

5 PREVENTION

Measures to control exposure to environmental carcinogens responsible for the occurrence of malignant neoplasm of the bladder related to work, among which are thetar, thearomatic amines and emissions from coke ovens are designed to eliminate occupational exposure or reduce it to levels approaching zero, through:

- entrapment and isolation procedures of work sectors;
- hygiene standards and strict safety measures for general cleaning of work environments, dehigiene staff, resources for bathing, washing hands, arms, face and exchange of clothing;
- local exhaust ventilation systems and general ventilation adequate and efficient;
- systematic monitoring of dust concentrations in ambient air;
- changes in work organization that allow reducing the number of exposed workers and exposure time;
- provision by the employer of appropriate personal protective equipment in good repair, as a complement to collective protection measures adopted.

The respiratory protective masks should be used as a temporary measure in emergências.Quando protection measures collective are insufficient, these should be carefully indicated for some sectors or functions. Workers must be trained appropriately for their use. Masks should be of quality and relevance to exposures with chemical or dust filters, specific for each substance handled or groups of substances which may be retained by the same filter. Filters should be changed strictly according to the manufacturer's recommendations. The Normative / MTb No. 1 / 1994 lays down rules on the use of technical equipment for respiratory protection. It is recommended to check the suitability and adoption by the employer of the control measures of occupational risk factors and health promotion identified in the PPRA (NR 9) and PCMSO (NR 7), plus thers regulations - health and environmental - in the states and municipalities. The NR 15 defines the LT concentrations in ambient air for days of 48 hours a week for several chemicals-related disease.

Annex 11 provides the LT 4 ppm or 15 mg/m³ aniline. Annex 13 provides for the prohibition of exposure or contact by any means, with the following substances or processes:

4-amino diphenyl (p-xenilamina); production benzidine; beta-naphthylamine and 4-nitrodiphenyl. These limits should be compared with those adopted by other countries and reviewed periodically in the light of knowledge and evidence

updated. It has been observed that even when strictly followed, do not prevent the appearance of damage to health.

The periodic medical examination aimed at identifying signs and symptoms for early detection of disease through clinical evaluation and complementary tests defined from the nature of occupational exposure. The IBMP for anilines are p-Aminophenol in the urine of 50 mg / g creatinine and 5% methemoglobin blood.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through the instruments themselves, the DRT / MTE and the union.

Epidemiological studies of risk assessment (risk assessment) conducted by OSHA in the United States, estimate that exposure to benzene at 10 parts per million (ppm) during the working life, produces an excess of 95 deaths leukemia in each 1,000 workers who were exposed.

With the same methodology, estimated that at concentrations of 1 ppm exposure during working life still causes an excess of 10 deaths leukemia per 1,000 workers. Other studies (Rinsky et al, 1987) show that workers occupationally exposed to benzene, in an average concentration of 10 ppm for 40 years, had an increased risk

to die for leukemia in 154 times. Lowering the exposure limit to 1 ppm, the risk would be 1, since July. At 0, 1 ppm, the risk would be virtually equivalent to the baseline risk of the exposed population.

The following etiologic agents and risk factors of occupational nature should be considered in

investigation of the cause of leukemia workers:

- benzene;
- ionizing radiation;
- EO;
- antineoplastic agents;

- electromagnetic fields (this is a controversial issue, there are studies that relate leukemia with exposure to electromagnetic fields and others who deny this relationship. There is no known experimental demonstration of this relationship and there are no known physical mechanism able to mediate a relationship between the agent and the cellular changes necessary for the onset of cancer);

- chlorinated pesticides (chlordane and heptachlor).

The leukemias - Mainly acute myeloid leukemia - Can be classified as doenças relacionadas to work, the Group II of the Classification of Schilling and the work is considered as a risk factor in the set of risk factors associated with multifactorial aetiology of these neoplasms.]

3 CLINICAL AND DIAGNOSTIC

The acute leukemias are characterized by:

- onset of clinical signs of abrupt and stormy: the majority of patients have this picture in the first 3 months from the onset of symptoms;

- symptoms related to depression of normal bone marrow function: fatigue due to anemia, fever due to infection due to the absence of mature leukocytes, bleeding (petechiae, ecchymosis, epistaxis, gingival bleeding, etc..) secondary to thrombocytopenia;

- generalized lymphadenopathy, splenomegaly and hepatomegaly resulting from infiltration by leukemic cells;

- bone marrow involvement with subperiosteal infiltration, resulting in bone pain sensation;

- leukemic infiltration of the meninges: may cause headache, vomiting, papilledema, cranial nerve palsies and other manifestations of central nervous system. May occur or subarachnoid hemorrhages.

In chronic myeloid leukemia, the initial symptoms are nonspecific and include fatigue, weakness, weight loss and anorexia. The large splenomegaly causes a feeling of fullness. After about 3 to 4 years, approximately 50% of patients enter a stage characterized by rapid increase in anemia, thrombocytopenia, and transform into acute leukemia (Blast crisis). In the remaining 50% of patients, the crisis blástica ocorre abruptly, without the intermediate stage of acceleration.

In chronic lymphocytic leukemia, often asymptomatic or without specific symptoms may appear, fatigue, weight loss and anorexia. Lymphadenopathy and hepatosplenomegaly are present in 50-60% of patients.

The diagnosis of acute leukemias is established by bone marrow

examination. The blast should be responsible for more than 30% of nucleated cells, to establish the diagnosis. Cytochemistry to be performed in all cases acute leukemia. The cell surface markers should be evaluated in all

suspected cases of acute lymphoblastic leukemia. In acute leukemia, anemia is almost always present. In about 50% of patients, the white cell count is below 10,000 cells per mm³ of blood, while

that about 20% of patients have counts exceeding 100,000 cells per mm³. The immature white blood cells, including blast forms, are found in circulating blood and bone marrow, where they represent 60 to 100% of all cells. Platelet counts show up, in 90% of cases, depressed, less than 50,000 per mm³.

In chronic lymphocytic leukemia (CLL), said not only clinical, anemia can be caused by lymphocytic infiltration of bone marrow by hypersplenism, autoimmune hemolysis and other causes. Absolute lymphocyte count ranges from 10,000 to 150,000/mm³, but may exceed 500,000 cells per mm³. Lymphocytes mature appearance, with little cytoplasm. The bone marrow examination is generally not necessary for diagnosis in patients with persistent lymphocytosis. The bone marrow of all patients with CLL contains at least 40% lymphocytes.

The demonstration of persistent lymphocytosis in patients within the age range of risk is enough to establish the diagnosis of CLL. Lymphocyte counts in excess of 15,000 per mm³, in patients over 50 years of age, are almost always the result of CLL.

In Chronic myeloid leukemia (CML) diagnosis is generally easily made based on a constellation of findings. No examination is pathognomonic of CML. The clinical picture evolves during a chronic phase to blast crisis of frequent acute leukemia. At the CBC, a normocytic normochromic-mild to moderate is usually observed. Granulocyte counts exceed 30,000 cells per mm³, reaching 100,000 to 300,000 per mm³ at the time of diagnosis. The peripheral blood smear is dramatic and represents an

deviation of cells for bone marrow out of a supersaturated. It is often described as that seems peripheral blood bone marrow. The granulocytes are normal in appearance and functionality. The more mature neutrophils elements are present in greater numbers. The myeloblasts and promyelocytes constitute less than 10% of leukocytes.

Unlike acute leukemia, the discontinuity in the granulocyte maturation is not present. About 50% of patients have thrombocytosis, which may exceed one million platelets per mm³. The spinal

bone has markedly hypercellular, resulting in a massive

granulocytic hyperplasia. The Philadelphia chromosome is found in about 90% of CML patients (peripheral blood and / or bone marrow).

S CONDUTA STRATAMENTO AND OTHER

The specialized treatment should be directed by a hematologist or oncologist. On the evolution of acute myeloid leukemia (AML), the most closely related to work, in particular to occupational exposure to benzene, one can say that the complete remission defines the prognosis. Complete remission is defined as follows:

- bone marrow containing less than 5% blasts;
- normalization of red cell counts, granulocyte and platelet counts;
- visceromegalies resolution;
- back to normal performance.

Patients who develop AML after a therapy-based cytotoxic agents, or who have other syndromes of bone marrow failure develops into a worse. Aspects of possible favorable prognosis include:

- WBC count less than 30,000 cells/mm³;
- rapid rate of reduction of leukemic cell therapy;
- serum LDH less than 400 IU;
- M2 subtype with t (8; 21) cytogenetic studies;
- subtype M4 with abnormal eosinophils in bone marrow.

The median survival for patients who get complete remission is 12 to 24 months. About 15 to 25% of patients achieving complete remission survive five years or more, and many of these patients can be cured. Most relapses occur within the first three years.

5. PREVENTION

Environmental surveillance of work-related leukemia must follow the provisions of the Convention / ILO No. 139/1974, which deals with the Prevention and Control of Occupational Hazards Caused by Carcinogenic Substances and Agents, presented in the introduction to this chapter.

Environmental control of exposure to benzene, ionizing radiation, the ethylene oxide chlorinated pesticide, and other agents, can effectively reduce the incidence of leukemias in exposed workers.

Malignant neoplasm of bone and articular cartilage of the members in this chapter. For monitoring of exposure to benzene and specific norms in force in Brazil ..

The control of exposure to EO should follow the technical standards established by the Interministerial / MS / MTb / MPAS No. 4 /

1991 concerning the handling, the register, the housing and the boundary conditions of operation and safety of the environment and personnel in sterilization units material, the process gas

EO or their mixtures with inert gas liquefied. The LT for exposure to EO is 1 ppm or 1, 8 mg/m³, air concentration for a normal working day of eight hours. The maximum permissible concentration of exposure in 15-minute period is 10 ppm. Federal Law No. 7.802/1989 and some state and local laws prohibit the use of organochlorine pesticide, including insecticides in them chlordane and heptachlor, and should not therefore be allowed its manufacturing

and marketing. Workers exposed to chlorinated pesticides should be monitored for effects arising from previous exposure. Exposure to electromagnetic fields should be monitored for signs and symptoms is still not sufficiently known. In any case, epidemiological studies should be conducted for further understanding of its effects on health.

Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT for Social Security as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

MALIGNANT NEOPLASMS OF THE LARYNX ICD-10 C32. -

1. DEFINITION OF THE DISEASE - DESCRIPTION

The malignant neoplasms of larynx cover three locations: the carcinoma of the glottis, or vocal cord true, the most common, representing about 57% of cases, thesupraglottic carcinoma, 35% of cases and the subglottic carcinoma, which corresponds to about 8% of cases.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The causes of laryngeal cancer are not well known. Among the risk factors described are smoking, alcohol consumption and exposure to excessive radiation, caused, for example, by a large amount of dental radiographs.

Risk factors of occupational nature, relatively well documented from epidemiological point of view, is exposure to strong inorganic acid mists, the asbestos or asbestos (Some results are controversial in literature, but there is a tendency to regard it as a carcinogen tumors of the larynx) occupational exposure to compounds nickel, the manufacturing process isopropyl alcohol, by the method of strong acid, to mustard gas and mineral oils (Soluble or cutting).

Regarding Asbestos, classical studies refer to mining workers, construction, shipyard and manufacturing of asbestos and asbestos cement. There seems to be clinical or pathological agents related to specific occupational nature.

The laryngeal cancer can be classified as work related disease, the Group II of the Classification of Schilling and the work, particularly exposure to occupational Asbestos is considered the set of risk factors associated with the etiology of this multifactorial tumor.

3. CLINICAL AND DIAGNOSTIC

Hoarseness is usually the first symptom appears. Patients with a history of hoarseness for more than three weeks duration should be carefully examined by laryngoscopy. May also be present otalgia, dysphagia, sore throat and cough. The diagnosis is based on a history of persistent hoarseness in patients over 40 years of age. This indirect laryngoscopy should be performed during speech, trying to observe the limited mobility of the vocal cords and arytenoids or stiffness. Computed tomography and / or magnetic resonance imaging of the larynx may be helpful. The differential diagnosis must be made with hyperkeratosis, laryngocele with polyps, pedunculated masses that appear as bright and papillomas, like formations that appear in clusters and white.

4. TREATMENT AND OTHER CONDUITS

In treatment, we seek to preserve both the life of the patient about his voice being used more limited surgical procedures, combined with radiotherapy or radiotherapy only, alone. Total laryngectomy is often required for those patients in whom more conservative methods have failed. The tumors deeply infiltrating are more difficult to assess due to the fact that they are accompanied by edema and distortion of the structures, and laryngectomy on these patients.

In general, the prognosis of laryngeal cancer, in terms of survival, is still relatively poor, depending on your location (glottic, supraglottic or subglottic), of early diagnosis, the degree of regional lymph node infiltration and presence of distant metastases. In the early stages, is

achieved by radiotherapy, cure approximately 75% of patients. In advanced stages, surgery and radiotherapy combined fail to hit more than 25% survival at 5 years.

This is shown, for specialized services to enhance the preservation of the voice of laryngectomized patients, searching for both processes, rehabilitation therapists, speech in order to develop esophageal speech. These services can be 50 to 70% of patients develop esophageal speech.

CHAPTER 12

Neoplasm (tumor)

RELATED WORK

(Group II of ICD-10)

7.1 INTRODUCTION

The term tumors or neoplasms designates a group of diseases characterized by loss of control of the process of cellular division, by which tissues normally grow and / or renew themselves, leading to uncontrolled cell multiplication. The lack of mechanisms to regulate and control cell proliferation, in addition to uncontrolled growth, can lead, in the case of cancer, invasion of neighboring tissues and spread to other parts of the body, producing metastasis.

Although not all known mechanisms, experimental studies have demonstrated that the cellular changes responsible for the production of tumor may originate in a single cell and involves two stages.

In the first, called initiation, irreversible changes (mutations) occur in the cell's genetic material. In the second stage, called the promotion, intra-and extracellular changes allow the proliferation of transformed cell, resulting in a nodule that in later stages, can spread to different regions of the body.

The oncogenesis may be triggered by environmental agents, acting on specific genes, leading to the onset and growth of tumors. Other suppressor genes function as regulating the proliferation of normal cells. The tumors are developed when this balance is disturbed by the influence of endogenous or genetic and / or exogenous and environmental. It is considered that the process of carcinogenesis is multifactorial. Among the factors involved are genetic predisposition or induced by secondary factors, environmental or viral infections. Ruptured defense mechanisms after a variable time, the precancerous lesion becomes a tumor malignant, invasive.

The cancer may arise as a result of exposure to carcinogens in the environment where he lives and works resulting from lifestyle and environmental factors produced or altered by human activity. According to data from the National Cancer Institute (INCA, 1995), estimate that 60 to 90% of cancers are due to exposure to environmental factors. In about 30% of cases, has not been possible to identify the cause of cancer, being attributed to genetic factors and spontaneous mutations. The wide variation observed in the international statistics on the incidence of cancer

strengthens the explanatory hypothesis that environmental factors attributed to the greater share of responsibility for disease.

Another important evidence concerns the observation that migrant populations are now present patterns of occurrence of

cancers similar to the adopted country. Must also be taken into account genetic differences between populations and facilities for diagnosis and disease registry.

The latency period is the time elapsed between the start of carcinogen exposure, which triggers the cellular changes and clinical detection of tumor. Has a variable duration, usually long, 20 to 50 years for solid tumors, or short, from 4 to 5 years for cancers of the blood. The long latency periods make it difficult to establish causal correlation or nexus between exposure and disease, particularly in the case of cancers

trabalho. Nos related to the developed countries that have reliable statistics, the cancer constitutes the second cause of death in adults, accounting for one in five deaths. The information available on the prevalence of cancer in Brazil are poor and do not reflect reality. The disease is the second leading cause of death in the Brazilian population over 40 years, the lung cancer is prevalent among men.

Among the malignancies prevalent and deadly in Brazil are the breast, cervix, stomach, lung, colon / rectum, prostate and esophagus. Mostly the result of direct aggression of the external environmental factors or stimuli

hormonal constant, which can be prevented or detected and successfully treated in early stages.

Regarding the causative agents of cancer, in general, the information is based on epidemiological studies in animals and in vitro

- occupational cancers do not differ in their morphological and histological characteristics, of other cancers;

- overall there is combined exposure and / or consequential. On the other hand, have in common with other occupational diseases to the difficulty of linking exposure to disease and the fact that are mostly preventable.

Thus, the effective oversight of occupational cancer is made about the processes and activities of working with carcinogenic potential, ie, risks or exposures. Surveillance of injuries or health effects seek early detection of cases and investigation of possible working relationship with the metering to identify and control intervention.

The health surveillance in relation to cancers related to work, basically consists in monitoring the environment and working conditions

and monitoring of the effects or damage to health. It is based on clinical knowledge, epidemiologic, industrial hygiene, ergonomics, toxicology, psychology, among other subjects, the perception of workers about their work and health and technical standards and regulations.

As a general guideline, environmental surveillance should follow the provisions of the Convention / ILO No. 139/1974, which includes:

- seek, in every way, replace carcinogenic substances and agents for other non-cancerous or less harmful;
- reduce the number of employees exposed, duration and exposure levels to a minimum consistent with safety;
- prescribe measures;
- establish appropriate system of record;
- inform workers about the risks and measures to be applied;
- ensure that medical exams needed to evaluate the effects of exposure.

Measures to control exposure to environmental carcinogens aim to:

- maintaining concentration levels approaching zero;
- entrapment and isolation procedures of work sectors;
- hygiene standards and strict safety measures and general cleaning of work environments, clean with a damp or water cleaning of environmental surfaces (countertops, walls, floors) or by suction, to remove particles prior to the commencement of activities;
- local exhaust ventilation systems and general ventilation adequate and efficient;
- systematic monitoring of concentrations of aerosols in ambient air;
- changes in work organization that allow reducing the number of exposed workers and exposure time;
- personal hygiene facilities, resources for bathing, washing hands, arms, face and exchange of clothing;
- provision by the employer of appropriate personal protective equipment in good repair in a complementary way to collective protection measures adopted.

STEPS AND ACTIONS FOR MONITORING ENVIRONMENTAL AND WORKING CONDITIONS - HEALTH SURVEILLANCE

Health services in public by those responsible for surveillance should be implemented

- mapping of productive activities, work processes and institutions in their territory or geographical area, developing and maintaining the records of records of establishments and the results of environmental assessments undertaken;

- classification of these activities and processes according to branch of economic activity (National Classification of Economic Activities);

- identification, in the catchment area, activities and processes demonstrably recognized as carcinogenic or probably carcinogenic to humans possibly through the comparison with information collected in the relevant literature, such as IARC, ACGIH and others;

- Sizing the population of employees included in these activities;

- survey of available data on health services, for example, time series of mortality cancer, high occurrence of leuconeutropenias laboratory tests, occurrence of rare cases, etc..;

- assessing the possible relationship of the cases identified in a geographic area with the establishments and existing work processes in the same area and / or occupational groups or categories of workers;

- Setting priorities for risk mapping in workplaces (industrial estates, agricultural industries, specific occupations, specific activity, etc.).

- estimation of human exposure levels, through technical studies of risk mapping and evaluation of work environments, in collaboration with universities, research institutes, Fundacentro, surveys and consulting firms;

- evaluation and revision of standards and regulations to protect health, identifying the possibilities of the ban and replacement substances with potential carcinogenic / genotoxic and recommending adoption of environmental control, engineering and occupational hygiene to decrease exposure levels;

- monitoring and evaluation of measures adopted.

7.3 STEPS AND ACTIONS FOR MONITORING OF EFFECTS ON HEALTH - EPIDEMIOLOGICAL SURVEILLANCE

Health services in public by those responsible for surveillance should be implemented

- construction and monitoring of the historical series of mortality occupational cancer and work-related, in the geographic area;

- identification of specific types of cancer more prevalent and / or recognized by the scientific literature as related to occupational exposures;

- type definition cancer or activities / occupations priority for surveillance purposes;

- incidences of cancer identified as priorities for monitoring, treated at hospitals and reported by SIH / SUS, the network of health services, oncology, hematology, specialized therapies and pathology laboratories. Assessment and monitoring of cases of Cancer Registry Population-Based;

- epidemiological investigation of cases of cancer identified as priorities for surveillance, trying to see the complete occupational history, and identify potential exposures associated intervention needs;

- epidemiological studies, particularly case-control research and surveys with cytogenetic alterations in selected groups.

It discusses, in actuality, the validity of the use of markers of exposure and identification of individuals susceptible to certain types of cancer, which can be applied in preemployment examinations of workers.

Among these, tests have been studied for chromosomal abnormalities and the presence of enzymes and proteins.

However, still no irrefutable scientific criteria are available for your use. The main difficulties

due to:

- low specificity of the tests - high rate of false-positive tests;
- invasive nature of some methods;
- little effect on mortality rates;
- high cost of some of the tests;
- non-validating tests and enzymatic markers, depending on research

Additional;

- non-consolidated scientific knowledge;
- ethical issues, with the possibility of discrimination of susceptible, preemployment examinations, as opposed to the need to control and eliminate exposure.

7.4 PROCEDURES AND CONDUCT TO BE TAKEN IF THE CASE OF DETECTING CANCER IN A DATA ESTABLISHMENT OF WORK *

Each case of work-related cancer should be confirmed or refuted through the following

following:

- establish histological type, date of diagnosis, demographics, age and sex;
- stratify the company's workers by sex and age;

CHAPTER 13

DISEASES OF BLOOD AND BODIES RELATED HEMATOPOIETIC THE WORK

(GROUP III OF ICD-10)

8.1 INTRODUCTION

The hematopoietic system is a complex formed by the bone marrow and other organs and blood hemoformadores. In the bone marrow are produced continuously, blood cells: red cells, neutrophils and platelets, under strict control of growth factors. To fulfill their physiological function, the cellular elements of blood must flow in both number and structure protection.

The productive capacity of bone marrow is impressive. Daily, she replaces three billion red cells per kilogram of body weight. Neutrophils have a half-life of only about 6 hours and 1, 6 billion neutrophils per kilogram of body weight need to be produced each day. An entire population of platelets should be replaced every 10 days. All this intense bone marrow makes too sensitive to infection, chemical agents, the metabolic and environmental factors that affect DNA synthesis or cell formation.

And also, therefore, the examination of peripheral blood proves a sensitive and accurate mirror of spinal cord activity.

In adult humans, the main hematopoietic organ located in the medullar bone of the sternum, ribs, vertebrae and iliac. The bone marrow is formed by a stroma cells and hemoformadoras originated in primitive multipotent cells (stem cell). This primitive cell is divided initially in primary lymphoid cells and primary myeloid cells from three strains. Under the control of offending drugs, these primordial cells undergo a process of differentiation and proliferation, giving rise, after the formation of precursors, the cells circulating in peripheral blood.

The offending drugs have specific for different strains of células. Entre cite the best known is the erythropoietin, the thrombopoietin and granulocytin (Growth factor granulocyte colony [G-CSF] and growth factor granulocyte colony-macrophage [GM-CSF]). Some of these substances have been produced and tested, and promising therapeutic weapons. The erythropoietin has already been used successfully for some indications clinics.

Blood cells, after reaching maturity, come into the sinusoids of the

marrow and reach the bloodstream, where they will perform their duties. Other important forming organs are the thymus tissue, lymph nodes and spleen, where lymphocyte development occurs, the processing antigense antibody production.

Assaults on the hematopoietic system may occur in the bone marrow, affecting the cell or primitive multipotent cells derived therefrom and into the bloodstream, destroying or altering the function of cells already formed.

Among the agents haematotoxic of interest to the health of the worker stand out from the benzene and ionizing radiation. These agents can damage the primitive multipotent cells, reducing their number or causing cytogenetic damage, resulting in hipoprodução cell or abnormal cell lines.

The functioning of the hematopoietic system can be evaluated by clinical history and results of physical examinations and laboratory. A detailed occupational history which allows to establish the nexus of a possible dysfunction and / or illness with work. The limit values of circulating blood cells, considered normal in the international literature, and some indices of clinical significance, are presented in Table XV. Não there is unanimity about the values considered normais. A analysis of these values must take into account the interindividual variations, such as age, sex, ethnicity, race, altitude, ambient temperature, socioeconomic conditions and intra-individual, such as time of day, physical exercise smoking, drug use, among other factors. They exhibit a Gaussian distribution in the population, where 2, 5% of normal will be below and above these limits.

The interpretation of the historical series is the best way to evaluate a discrete changes in blood and which is not sudden. The term anemia of any cause or mechanism, which often is referred to, means a decrease

The number of red blood cells and / or hemoglobin and / or hematocrit values compared to the normal range. It is important to examine also the volume of red blood cells and hemoglobin concentration, classifying the anemia in micro-or hypo-or macrocytic and normochromic. The information regarding the volume and concentration of hemoglobin in the RBC are obtained indirectly by calculating the mean corpuscular volume (MCV) and hemoglobin concentration corpuscular concentration (MCHC).

The reticulocyte count, which is the young red cell, between 24-36 hours after leaving the marrow, or reticulocyte index, reflects the dynamics of erythropoiesis, ie, the ability of bone renewal of red blood

cells circulating. Leukocytes should be analyzed in absolute numbers of specific cells and not in global terms and percentages. Microscopic examination of blood smear, or hematoscopia also contributes to the morphological analysis of cells.

The bone marrow examination can be performed through the analysis of material obtained from aspiration or biopsy. The aspirate allows examination of cell morphology and the establishment of reason myeloid / erythroid ratio (M / E).

The biopsy shows the cellularity of the sample obtained and is particularly useful in cases of marrow infiltration (lymphoma and other carcinomas) and leukemia.

The examinations for evaluation of the coagulation system, is quoted in regard to specific diseases such as purpura and other hemorrhagic manifestations. Some specific clinical picture may require more sophisticated tests such as hemoglobin electrophoresis and the chromosomal and cytogenetic analysis.

The prevention of diseases of blood and blood forming organs related to work nosprocedimentos based health surveillance of workers: surveillance of environments and working conditions and damage to health surveillance. Uses knowledge of clinical epidemiology, industrial hygiene, toxicology, ergonomics and psychology, among other disciplines, as well as workers' perceptions about their work and health and technical standards and regulations. These procedures can be summarized as follows:

- reconnaissance activities and workplaces where there are chemicals, physical agents, and / or biological risk factors arising from work organization, potentially causing disease;
- identification of problems or damage to health, potential or actual, arising from exposure to identified risk factors;
- identifying and proposing measures to control that must be taken to eliminate or control exposure to risk factors and for the protection of workers;
- education and information to workers and employers.

Upon confirmation of the diagnosis of disease and its relationship to work, following the procedures described in Chapter 2, the health services responsible for the care worker should program the following

Actions:

- assessing the need for removal (temporary or permanent) worker exposure, sector of employment or work as a whole;
- whether the worker is insured by the Social Security SAT, request the issuance of CAT to the company, fill in the LEM and forward to the

INSS. In case of refusal to issue the CAT by the company, the doctor (or medical services) should do so;

- monitoring of progress, and worsening record of clinical worsening and their relationship to return to work;

- notification of this disease to the information system of morbidity of the SUS, the DRT and the labor union of workers;

- implement the epidemiological surveillance activities aiming at identifying the occurrence of the disease, through the active search for other cases in the same company or workplace or other companies of same industry in the geographic area;

- if necessary, supplement the identification of the agent (chemical, physical or biological) and working conditions determine the disorder and other risk factors that may be contributing to the occurrence;

- inspection at the company or work environment where the patient or worked in other companies of same industry in the geographic area, identifying risk factors for health, measures of collective protection, equipment and personal protective measures used;

- identification and recommendation to the employer regarding the protection measures and recommendations to be adopted, informing the workers.

The measures of protection and prevention of exposure to risk factors at work include:

- replacement of production technologies by less hazardous to health;

- isolation of the agent / substance or enclosing the process;

- strict measures of hygiene and safety at work, for example, adoption of local exhaust ventilation systems and appropriate general and efficient, use of exhaust fume, control of leaks and incidents through preventive and corrective maintenance of machinery and equipment and monitoring of compliance;

- environmental monitoring and systematic adoption of systems insurance work, operational and transportation, sorting and labeling of chemical substances toxicological properties and toxicity;

- information and risk communication to workers;

- maintenance of general environmental conditions and comfort for employees and adequate facilities for personal hygiene and adequate sanitary facilities, restrooms, showers, sinks with clean running water and an abundance of clothing and cleaned daily;

- decrease the exposure time and the number of exposed workers;

- provision of individual protection equipment and proper maintenance indicated in a complementary way to measures of collective

protection.

In the case of benzene, which has a well-known myelotoxic action should be followed the guidelines of the Ordinance / MTb No. 14/1995 and Instruction / MTb No. 1 / 1995, which define the methodology for assessing concentrations of benzene in work environments and the development of Prevention Programme **Exhibition Occupational Benzene (PPEOB) by the employer, processors and users of benzene.**

The Reference Value Technology (VRT) established for the benzene is 1, 0 ppm for the companies mentioned in Annex No. 13-A, and de 2, 5 ppm for steelmakers. The companies producing anhydrous alcohol should replace benzene. A Interministerial / MS / MTb No. 3 / 1982 banned in all national territory to manufacture products

containing benzene in its composition, admitting, however, the presence of this substance as a contaminant with a percentage not exceeding 1% by volume. Thus, from the late '80s, the presence of benzene in the solvents used in paints, varnishes, thinners, glues, etc.. have not been found, except in trace levels. The price of petrol still contains a content benzene which can range from 0, 5-3% depending on the type and manner of production and thus the risk of fuel also offers leukemia. One should also note that other oil products like kerosene, liquefied petroleum gas (LPG), diesel oil, fuel oil, lubricating oils, among others, have no benzene, except in negligible amounts in occupational terms. Despite this, it is recommended to maintain regular surveillance by the requirement that producers can prove the contents of benzene in their finished products.

Exposure to ionizing radiation should be limited to the control of radiation sources, both in industrial environments and in health services. Observe the Basic Guidelines on Radiological Protection in Medical and Dental Radiology, defined by Ordinance / MS No. 453/1998.

The equipment should have safety devices, protective shields and undergo rigorous preventive maintenance, the rooms and areas shall be provided with signage, and screen, operating procedures and safety must be well defined, including accident and emergency situations, the Personnel should receive adequate training and be supervised, equipment and supplies must be positioned as far as possible from workers, it must be flushed down the number of workers in these sectors and the exposure time.

Federal Law No. 7.802/1989 and some state and local laws prohibit the use of organochlorine pesticides and should not therefore be allowed for manufacturing and marketing. Other groups have also prohibited its production, sales, use, transportation and destination

defined by this law.

Some states and municipalities have regulations still to be obeyed. It is recommended to observe compliance by the employer, Regulating Rural (NRR) Ordinance / MTb No. 3067 / 1988, especially the NRR 5, which deals with chemicals and pesticides, fertilizers. Special attention should be given to the protection of workers involved in the activities of preparation of grout and

application of these products. The NR 7 and 15 of the Ordinance / No MTb 3.214/1978, should be consulted because they define the parameters for the surveillance of the damage or effects on workers' health in Brazil.

**LIST OF DISEASES OF BLOOD AND HEMATOPOIETIC
ORGANS RELATED TO THE WORK,
PURSUANT TO ORDINANCE / MS No. 1.339/1999**

- Myelodysplastic syndromes (D46. -)
 - Other anemias due to enzyme disorders (D55. 8)
 - Acquired hemolytic anemia (D59. -)
 - Aplastic anemia due to other external agents (D61. two) and aplastic anemia unspecified (D61. nine)
 - Purpura and other hemorrhagic manifestations (D69. -)
 - Agranulocytosis (neutropenia toxic) (D70)
 - Other specified disorders of white blood cells, leukocytosis, leukemoid reaction (D72. 8)
 - Methemoglobinemia (D74. -)
- 8.3.1 Myelodysplastic Syndrome ICD-10 D46 .-

1. DEFINITION OF THE DISEASE - DESCRIPTION

The myelodysplastic syndromes (MDS) are a group of acquired clonal disorders of hematopoietic cell paramount. They are characterized by cytopenia, hypercellular marrow and various morphological abnormalities. Despite the presence of adequate number of primary hematopoietic cells, is ineffective hematopoiesis resulting in multiple cytopenias. Entities are chronic, variable duration and may precede the onset of acute myelogenous leukemia, which can occur in 10 to 40% of cases, MDS called by some, as pre-leukemia.

**2. EPIDEMIOLOGY - RISK FACTORS OF
OCCUPATIONAL NATURE KNOWN**

The myelodysplastic syndromes are usually considered idiopathic, but have been observed after chemotherapy, especially procarbazine for

disease Hodgkingemelphalan for multiple myeloma or carcinoma of the ovary. Also the chloramphenicol, thecolchicine and the nitrous oxide have been related to these syndromes, as well as the use of nonsteroidal antiinflammatory drugs, such as phenylbutazone.

Some predisposing factors for SMD are genetic. In some patients there is weakness or inability to chromosomal DNA in repairing the effects suffered after exposure to ionizing radiation. Consequences occur as aberrations in the DNA that stimulate certain oncogenes. You should carry out monitoring of occupational exposure or environmental alkylating agents to people, phenylbutazone, insecticides, pesticides and organic solvents.

Occupational exposure to benzene and ionizing radiation show a causal association with the development of myelodysplastic syndromes.

In the case of benzene, the myelodysplasia are linked to exposure to relatively high concentrations. In actuality, should be valued exposure to benzene in petrochemical and chemical industries, laboratory and large steel mills and coke plants which have units attached carbochemical in general. At low levels, sometimes traits, there may be exposure to benzene by the use of solvents in paints, varnishes, thinners, removers, degreasers, adhesives and kerosene. In the handling of gasoline is the small probability of occurrence of myelodysplasia because of low levels of benzene (0, 8-3%) contained in the fuel. It is important to note that a value of 3% benzene, into finished products, exceeding the limit determined by the Interministerial / MS / MTb No. 3 / 1982.

Besides benzene, various other substances may be linked to myelodysplasia, such as arsenic compounds and the ethylene oxide. Probably associated with other substances myelodysplasia are:

- solvents 2-ethoxyethanol and the 2-methoxyethanol (Glycol ethers);

- TNT (explosive);

- dinitrophenol;

- pentachlorophenol (PCP, also known in Brazil as a powder from China);

- hexachlorocyclohexane (HCH, or lindane, also popularly known as BHC);

- p-hydroquinone (Solid minor occupational, and may be one of the metabolites benzene myelotoxicity responsible for this product Styrene (monomer of polystyrene), 2 - butoxyethanol (glycol ether), chlorobenzene, dichlorobenzene and organochlorine pesticides, dieldrin and the heptachlor are still loosely associated with myelodysplasia.

In workers exposed to these chemicals, in which other non-occupational causes of MDS were excluded, they can be classified as work-related diseases, the Group I of the Classification of Schilling in the work, particularly the occupational exposure to benzene and radiation ionizing, can be regarded as a necessary cause. It is unlikely that the disease occurs in the absence of this condition.

3. CLINICAL AND DIAGNOSTIC

The symptoms are related to the presence of anemia, insidious onset, which may later be followed by bleeding (secondary to thrombocytopenia) and infections (secondary to leukopenia). Most patients die when there is progression to pancytopenia.

The CBC shows cytopenia (isolated or multiple). The show can hematoscopia hipogranulados neutrophils, blasts and leukemic hiatus. The morphology of erythrocytes may be normal or even show hypochromic macrocytic giants. Platelets are generally large and grainy. The marrow is usually cellular, may, however, present hyper- or hypocellularity. There may be underdevelopment granulocytic,

bilobulated dwarf megakaryocytes, ringed sideroblasts and multinucleated giant normoblasts.

4. TREATMENT AND OTHER CONDUITS

Treatment aims to correct the cytopenias. The use of androgens (danazol, fluoxymesterone) have shown conflicting results. Some studies suggest significant association of anemia with erythropoietin and granulokinins. Marrow transplantation is a treatment option.

Thymelodysplasia evolves, usually to death. About 60 to 80% of patients die due to complications, for example, acute infection, hemorrhage or associated diseases.

About 10 to 20% are stable and they die from causes unrelated to the disease. In the case of benzene and ionizing radiation, the risk of transformation to Acute myelogenous leukemia depends on the percentage of blasts in the bone marrow.

Patients with refractory anemia can survive many years, and the risk of leukemia is low (<10%). Those with excess blasts or chronic myelogenous leukemia have short survival, usually less than 2 years, and are at higher risk (20 to 50%) of developing acute leukemia. Allogeneic transplants of bone marrow is the only definitive therapy, although it is difficult to determine the best time, given the broad spectrum of possibilities for prognosis.

Staging in myelodysplastic syndromes intertwined with the

concepts of evolution and prognosis. Can be used the criteria proposed for anemia and / or diseases of white blood cells. Criteria used for the staging of the deficiencies resulting in anemia are shown in Table XVI.

Once installed the SMD should be avoided further exposure to harmful agents and must accompany the patient to the risks of bleeding, infections and blast transformation.

5. PREVENTION

The prevention of SMD work-related basically consists in monitoring environments, working conditions and the effects or health hazards, as described in the introduction to this chapter. Environmental control of benzene and ionizing radiation can effectively reduce the incidence of disease in exposed workers. It is recommended to observe the adequacy of PPRA (NR 9) and PCMSO (NR 7) and compliance by the company, and other regulations - health and environmental - in the states and municipalities.

Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
 - examine the exposed, to identify other cases;
 - notify the case to the information systems of the SUS, the DRT / MTE and the labor union;
 - provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control of risk factors.

The procedures for monitoring of exposure to benzene and specific norms in force in Brazil are described in the protocol Aplastic anemia due to other external agents (8.3.4) this chapter, and for exposure to ionizing radiation, the protocol Malignant neoplasm of bone and articular cartilage of the members (7.6.7)

OTHER ENZYME DISORDERS anemia due to ICD-10 D55. 8

1. DEFINITION OF THE DISEASE - DESCRIPTION

The anemia is characterized by reducing the amount of functional hemoglobin circulating total. How in practice does not take into account possible variations in blood volume, the anemia usually defined as a reduction of hemoglobin concentration in peripheral blood below 13 g/100 ml in men, or 11 g/100 ml in women. Anemia by enzymatic disorders are those caused by defects in production of red blood cells, particularly in the synthesis of hemoglobin.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Lead is a classic example of agent that interferes with heme synthesis dogrupo hemoglobin by interfering with enzyme systems such as the ALA-dehydratase, the coproporphyrinogenase and heme synthetase.

Lead also causes hemolysis (see Acquired Hemolytic Anemia).

Other examples of toxic substances present in work environments, which can interfere in the synthesis and biotransformation of heme, include:

- hexachlorobenzene (HCB);
- 2, 4-dichlorophenol (2, 4-D) and 2, 4, 5-Trichlorophenol (2, 4, 5-T) - widely used herbicides in agriculture known as Tordon, among others;
- tetrachlorodibenzo-p-dioxin (PCBs) - a contaminant of various products can be found in mixtures of 2, 4-dichlorophenol (2, 4-D) and 2, 4, 5-Trichlorophenol (2 4, 5-T);
- o-benzyl-p-chlorophenol;
- 2-benzyl-4, 6-dichlorophenol;
- vinyl chloride.

In exposed workers, in which other causes of anemia by enzymatic disorders nãoocupacionais were excluded, they can be classified as work-related diseases, the Group I of the Classification of Schilling in the work, particularly the occupational exposure to Lead and chlorophenol can be considered as a necessary cause. It is unlikely that the disease occurs in their absence.

3. CLINICAL AND DIAGNOSTIC

The anemia produced by Lead is just one of many manifestations of the clinical picture of chronic poisoning by this metal, including, abdominal pain, nephropathy, hypertension, abnormal sperm, peripheral neuropathy and encephalopathy. In adults, anemia (And its symptoms) can be observed with levels Lead blood over 50 g/100 ml.

The establishment of the connection between work, in cases secondary to exposure to lead, based on exposure history and laboratory confirmation by plasma levels of Lead blood or urine. According to NR 7, the dosage of VR Lead in blood (Pb-S) is 40 g/100 ml and the IBMP is 60g/100 ml, while mean excessive exposure, consistent with adverse effects on worker health. The ACGIH, the United States, recommends as biological exposure index value of 30g/100 ml. Other laboratory findings are the strengths in the urine of delta-aminolevulinic acid (ALA-U), with

VR in Brazil is currently at 4, 5 mg / g creatinine and the IBMP is 10 mg / g creatinine. For zinc protoporphyrin in blood (ZPP-S), VR is 40 g/100 ml and the IBMP is 100g/100 ml.

CBC shows anemia hypochromic and microcytic with reticulocytosis and the presence of basophilic granules in red blood cells, larger than usual, ranging from 0, 25-2, 00 micrometres more frequent in large cells (macrocytic), round or ovoid or as diplococci, varying in number (up to 10 or 20), and only rarely stained in blue. The arrangement of the grains becomes uniformly, sometimes concentrated in

point, or arranged like a crown on the periphery of the globule.

As a result of inhibition of heme, an accumulation of iron within the erythroblasts with training and siderocytes sideroblasts, which can be detected by staining with the dye of Prussia (blue positive) in the examination of material obtained by aspiration / biopsy of bone marrow.

4. TREATMENT AND OTHER CONDUITS

The most important therapeutic measure is the cessation of exposure. The anemia can be corrected by specific chelation therapy. Severe cases may require transfusion of red blood cells.

In poisoning Lead should be considered blood lead levels and the possibility that these blood levels may be causing injury and possible disability or dysfunction in other organs, equipment, systems or cell types.

For the staging of the disability caused by anemia, may be used as a reference, the parameters proposed by the American Medical Association (AMA) at its Guides to the Evaluation of Permanent Impairment (1995), presented in the previous protocol.

5. PREVENTION

The prevention of anemias due to enzyme disorders related to work basically consists in monitoring the environment and working conditions and monitoring of the effects or health hazards, as described in the introduction to this chapter. Environmental control of exposure to lead, hexachlorobenzene (HCB), herbicides 2, 4-dichlorophenol (2, 4-D) and 2, 4, 5-trichlorophenol (2, 4, 5-T), tetrachlorodibenzo-p-dioxin (dioxin), the benzyl- -p-chlorophenol, 2-benzyl-

4, 6-dichlorophenol, vinyl chloride and other causal agents can effectively reduce the incidence of disease in occupational groups at risk.

The environmental control measures aimed at elimination of exposure or to maintain them in concentration levels approaching zero, through:

- entrapment and isolation procedures of work sectors;
- use of hermetically sealed systems, industry
- adoption of standards of hygiene and strict security, with adequate exhaust ventilation systems and efficient;
- systematic monitoring of concentrations of agents in the air environment;
- changes in work organization that allow reducing the number of exposed workers and exposure time;
- measures of general cleaning of work environments and facilities for personal hygiene, as resources for bathing, washing hands, arms, face and exchange of clothing;
- provision by the employer of appropriate personal protective equipment in good repair, as indicated in a complementary way to measures of collective protection.

The respiratory protective masks should be used as a temporary measure in emergencies.

When the measures of collective protection is inadequate, they should be carefully indicated for some sectors or functions. Workers must be trained appropriately for their use. Masks should be of quality and relevance to exposures with chemical or dust filters specific for each substance handled or groups of substances which may be retained by the same filter. Filters should be changed strictly according to the manufacturer's recommendations. The Normative / MTb No. 1 / 1994 lays down rules on the use of technical equipment for respiratory protection.

It is recommended to check the adequacy of and compliance with PPRA (NR 9), the PCMSO (NR 7) and other regulations - health and environmental - in the states and municipalities. The NR 15 defines the LT concentrations in ambient air of various chemicals, for journeys of 48 hours a week.

The periodic medical examination aimed at identifying signs and symptoms for early detection of disease. In addition to thorough clinical examination, we recommend the use of standardized instruments and laboratory testing appropriate to the risk factor identified.

The procedures for health surveillance of workers exposed to vinyl chloride are described in the protocol Angiosarcoma of liver (7.6.2) in Chapter 7. In relation to lead exposure, see the protocol Lead colic (16.3.6). For exposure to benzene, see the protocol Aplastic anemia due to other external agents (8.3.4) this chapter.

Suspected or confirmed the relationship of disease with the work,

you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

Acquired hemolytic anemia ICD-10 D59. 2

1. DEFINITION OF THE DISEASE - DESCRIPTION

The anemia characterized by reducing the amount of functional hemoglobin circulating total. How in practice does not take into account possible variations in blood volume, the anemia usually defined as the reduction of hemoglobin concentration in peripheral blood below 13 g/100 ml in men, or 11 g/100 ml in women. Acquired hemolytic anemia is secondary anemia to decreased survival or destruction of erythrocytes associated with an inability of the bone marrow to compensate for the decrease in survival or destruction.

The pathophysiologic mechanism of hemolysis caused by toxic substances is still not fully understood. It seems to be due to exposure of sulfhydryl groups of red blood cell membrane and its connection with radicals and toxic substances, forming compounds that alter the permeability of the membrane, allowing passage

water and cations in the opposite direction of its concentration gradient, resulting in destruction of blood cells

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The decreased survival and destruction of blood cells occurs by the action of toxic substances, infectious agents, antibodies, and physical trauma. Among the toxic substances that may be present in environments works are:

- nitro and amino derivatives of benzene;
- arsine;
- lead;
- mercury;
- copper;
- manganese.

In exposed workers, in which other non-occupational causes of acquired hemolytic anemia were excluded, it can be classified as work related disease, the Group I of the Classification of Schilling in the work, particularly in occupational exposure to derivatives of amino benzene, thearsine, to lead,mercury,Copper and manganese, can be considered as a necessary cause.

CLINICAL AND DIAGNOSTIC

The clinical picture is characterized by anemia accompanied by jaundice due to increased serum concentration of indirect bilirubin, liver failure by conjugating bilirubin resulting from the metabolism of heme released from the hemoglobin molecule.

The anemia is of type normochromic with reticulocytosis. The hematocrit may show morphological changes of the erythrocyte, with poikilocytosis, polychromasia, fragmented red cells and remnants of cell membrane. The spinal osseapode show hyperplasia of the erythroid series, accompanied sometimes by an increase in other series and with effect from mild peripheral increased number of platelets.

Dosages of free hemoglobin in plasma indirect bilirubin and lactate dehydrogenase (LDH) are increased. The Coombs test is negative. The secondary cases with occupational exposure to agents such as arsine, theLead and the mercury can be confirmed by serum or urine of these agents.

4. TREATMENT AND OTHER CONDUITS

Supportive treatment with transfusions of concentrated blood cells in severe cases associated with intravenous hydration and urine alkalinization to decrease the precipitation of hemoglobin in renal tubules.

5. PREVENTION

The prevention of acquired hemolytic anemia related to work based on monitoring of environments, working conditions and the effects or health hazards,as described in the introduction to this chapter.

Environmental control of lead, and amino nitro-derivatives of benzene, arsine, mercury, copper and manganese may reduce the incidence of disease in occupational groups at risk. Control measures environmental seek elimination or reduction of exposure to concentrations close to zero, by:

- entrapment and isolation procedures of work sectors;

- use in industry, hermetically sealed systems;
- hygiene standards and strict security, with adoption of exhaust ventilation systems adequate and efficient;
- systematic monitoring of concentrations of agents in ambient air;
- changes in work organization that allow reducing the number of exposed workers and exposure time;
- measures of general cleaning of work environments and facilities for personal hygiene, as resources for bathing, washing hands, arms, face and exchange of clothing;
- provision by the employer of appropriate personal protective equipment in good repair, as indicated in a complementary way to measures of collective protection.

In mining, should be added:

- Wet drilling techniques to reduce dust concentrations in ambient air;
- use of respiratory protective masks, and if the levels are above acceptable, may be necessary to use air purifying equipment;
- Wet cleaning or water cleaning of environmental surfaces (countertops, walls and floor) or by suction, to remove particulates before the start of activities.

The respiratory protective masks should be used as a temporary measure in emergencies.

When the measures of collective protection is inadequate, they should be carefully indicated for some sectors or functions. Workers must be trained appropriately for their use. Masks should be of quality and relevance to exposures with chemical or dust filters, specific for each substance handled or groups of substances which may be retained by the same filter. Filters should be changed strictly according to the manufacturer's recommendations. The Normative / MTb No. 1 / 1994 lays down rules on the use of technical equipment for respiratory protection.

In the case of mercury, the floors and surfaces should be smooth with no edges or ridges, and should be adopted in soil drainage systems and metal bars on channels with water, to collect particles and spills, removing them immediately from the environment and avoiding its volatilization. Recommend control measures to prevent environmental contamination effluent water and soil.

It is recommended to check the adequacy of and compliance with PPRA (NR 9), the PCMSO (NR 7) and other regulations - health and environmental - in the states and municipalities. The NR 15 defines the LT concentrations in ambient air of various chemicals, for journeys of 48 hours per week of work,

example:

- Arsine: 0, 04 or 0 ppm, 16 mg/m³;
- Lead: 0, 1 mg/m³;
- Mercury: 0, 04 mg/m³.

For manganese, Ordinance / MTb No. 8 / 1992 establishes the LT up to 5 mg/m³ in air for journeys of up to 8 hours daily for mining operations, processing, milling, ore transport and other operations with exposure to dustmanganese or its compounds. For exposure to fumes manganese or compounds, the LT is up to 1 mg/m³ in air, to shift up to 8 hours / day.

The periodic medical examination aimed at identifying signs and symptoms for early detection of disease. In addition to thorough clinical examination, we recommend the use of standardized instruments and laboratory testing in accordance with the identified risk factors

For lead are adopted the following parameters:

- concentration Lead in blood (Pb-S) - VR up to 40 g/100 ml and 60-IBMPg/100 ml. (The ACGIH recommends biological index of exposure 30g/100 ml);
- amino acid concentration delta Levulinic in urine (ALA-U) - VR up to 4, 5 mg / g creatinine IBMP and up to 10 mg / g creatinine;
- zincoprotoporfirina concentration in blood (ZPP-S) - VR up to 40 g/100 ml and 100-IBMPg/100 ml.

The dosage of Lead serum reflects the absorption of the metal in the weeks preceding the sample collection or deposit mobilization of bone.

There biológico for monitoring occupational exposure to arsine, themanganese and copper.

The procedures for health surveillance of workers exposed to leadare described in the protocol Lead colic, Chapter 16. For exposure tobenzene, see item 5 of the ProtocolAplastic anemia due to other external agents, this chapter.

Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;

- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

Whereas 10-50% of cases of aplastic anemia are labeled as idiopathic, it is possible that occupational history taking, properly exploited, can help to clarify a possible link with the work.

Among the drugs, medicines and haematotoxic aplasiantes marrow, are well known: chloramphenicol, phenylbutazone, gold salts, sulfonamides, phenytoin, carbamazepine, tolbutamide and quinacrine.

The occupational etiology has been described in workers exposed to benzene, ionizing radiation, and less obvious:

- to arsenic compounds;
- to EO;
- to 2-ethoxyethanol;
- to 2-methoxyethanol;
- the TNT;
- to organochlorines such as pentachlorophenol (PCP, also known in Brazil as a powder from China) and hexachlorocyclohexane (HCH or Lindane, also known popularly BHC).

Exposure to high concentrations of benzene work environments (over 100, 200 ppm) resulted in the past, hundreds of cases of disease worldwide. In recent years, with the progressive reduction of ambient concentrations and improvement of working conditions, the occurrence of Secondary aplastic anemia exposure to benzene also reduced.

According to WHO, it is estimated that in occupationally exposed to benzene at 50 ppm by mperíodo a year, 5% developaplastic anemia. If exposed to 100 ppm during the same period, 10% of those exposed get sick. After 10 years of exposure to 10 ppm, 1% of those exposed would develop aplastic anemia; to 50 ppmexposure, 50% of those exposed would develop the disease, and in environments of 100 ppm benzene, 90% of patients would be exposed. Currently, these levels of exposure to benzene are difficult to observe, therefore, to be achieved, it would be necessary to work with direct exposure to benzene or mixture containing high proportions, more than 20% by volume.

In workers exposed under the conditions described above, in which other causes of aplastic anemia non-occupational, were excluded, it can be classified as work related disease, the Group I of the Classification of Schilling, since the job or occupation with exposure to benzene, ionizing radiation and / or other substances mentioned can be regarded as necessary causes.

3. CLINICAL AND DIAGNOSTIC

The clinical features are related to the low number of circulating blood cells. The onset is usually insidious but can be dramatic, depending on the severity and speed with which anemia evolves. The patient shows signs / symptoms anemia and may have bleeding secondary to thrombocytopenia and infections secondary to leukopenia. The spleen is not enlarged.

The diagnosis of aplastic anemia based on the association between peripheral cytopenia with characteristic empty marrow replaced by fat. The circulating red blood cells do not show abnormalities.

The differential diagnosis is made with myelodysplastic syndrome, hypersplenism, anemia secondary to marrow infiltration (leukemia, lymphoma) and severe sepsis.

The main criterion for classifying a severe marrow hypocellularity is, by observing less than 25% or even less than 50% of the marrow space with less than 30% of hematopoietic cells. The table also is serious when there are at least two of the following criteria:

- anemia with reticulocyte counts less than 40.000/mm³ or reticulocyte index below 1%;
- neutrophils less than 500/mm³;
- platelet count below 20,000 /

CHAPTER 14

Aplastic Anemia

TREATMENT AND OTHER CONDUITS

Treatment options available are the androgens, corticosteroids in high doses, antilymphocyte globulin, antithymocyte and bone marrow transplantation.

Transfusion of blood, red blood cells or platelet concentrates in patients candidates for bone marrow transplant should be performed only when absolutely necessary.

Staging in aplastic anemia intertwined with the concepts of evolution and prognosis. Can be used as reference parameters for evaluation and staging of the disability-related anemia, proposed by the AMA in Guides to the Evaluation of Permanent Impairment (4. Nd edition, 1995) shown in Table XVI in the previous protocol. In the case of intoxication benzene, primarily responsible for aplastic anemia related to work, one must take into account the possibility of further damage and the involvement of other organs, equipment, systems or cell types.

5. PREVENTION

The prevention of aplastic anemia related to work due to other external agents based on monitoring of environments, working conditions and the effects or health hazards, as described in the introduction to this chapter. Environmental control of exposure benzene, to ionizing radiation to pesticides and chlorinated inorganic arsenic may reduce the incidence of disease in occupational groups at risk. The environmental control measures aimed at the elimination or reduction of exposure to concentrations close to zero, by:

- entrapment and isolation procedures of work sectors;
- use in industry, hermetically sealed systems;
- hygiene standards and strict security, with adoption of exhaust ventilation systems adequate and efficient;
- systematic monitoring of concentrations of agents in ambient air;
- changes in work organization that allow reducing the number of exposed workers and exposure time;
- measures of general cleaning of work environments and facilities personal hygiene, such as resources for bathing, washing hands, arms, face and exchange of clothing;
- provision by the employer of appropriate personal protective

equipment in good repair, as indicated in a complementary way to measures of collective protection.

The respiratory protective masks should be used as a temporary measure in emergencies.

When the measures of collective protection is inadequate, they should be carefully indicated for some sectors or functions. Workers must be trained appropriately for their use. Masks should be of quality and relevance to exposures with chemical or dust filters, specific for each substance handled or groups of substances which may be retained by the same filter. Filters should be changed strictly according to the manufacturer's recommendations. The Normative / MTb No. 1 / 1994 lays down rules on the use of technical equipment for respiratory protection.

The Normative / MTb No. 2 / 1995 provides for the health surveillance of workers in the prevention of occupational exposure to benzene. This statement sets as tools for health surveillance in clinical and occupational history taking, physical examination, complementary examinations, comprising at least a complete blood count with platelet count and reticulocyte (it also sets the NR 7, every six months) ; the epidemiological and toxicological risk groups, obtained by evaluating biological indicators of exposure.

One of the biological indicators of exposure is the recommended concentration trans-trans muconic acid in urine, which reference value (RV) is 0, 5 mg / g creatinine, with a value of IBMP, 4 mg / g creatinine. The determination of the concentration of acid S-phenyl-mercaptopurine at the end of the working day is recommended by ACGIH

(2001). Your IBMP is 25 g / g creatinine.

The health surveillance of workers exposed to benzene should follow the guidelines of the Ordinance / MTb No. 14/1995 and Instruction / MTb No. 1 / 1995, which define the methodology for assessing concentrations of benzene in the workplace and require the development of PPEOB by the employer, processors and users of benzene. The VRT established for benzene is 1, 0 ppm for the companies covered in Appendix 13-A (with the exception of the steel companies, producing anhydrous alcohol and those who should replace benzene) and 2, 5 ppm for steelmakers.

OSHA establishes permissible exposure limit (PEL) for benzene 1 ppm (3, 2 mg/m³). The exposure limit (TLV-TWA) for benzene, adopted by ACGIH in 2001, is 0, 5 ppm (1, 6 mg/m³), with the observation that benzene should be considered confirmed human carcinogen, group A1. The limit for short exposures (STEL) proposed by the ACGIH is 2, 5 ppm (8 mg/m³). The recommended exposure limit (REL) established by

NIOSH is 0, 1 ppm, STEL for 15 minutes in the amount of 1 ppm.

Ordinance / MS / MTb No. 3 / 1982 banned throughout the country to manufacture products containing benzene in its composition, admitting, however, the presence of this substance as a contaminant with a percentage not to exceed 1% (one percent) by volume. Thus, from the late '80s, the presence of benzene in the solvents used in paints, varnishes, thinners, glues, etc.. has not been found except in trace levels. Gasoline also contains a contentbenzene which can range from 0, 5-3% depending on the type and form of production.

Thus, this fuel also offers a small risk of causing leukemia. Other petroleum products like LPG, kerosene, diesel oil, fuel oil and lubricating oils have no benzene except in quantities

negligible in occupational settings. Despite this, it is recommended to maintain regular surveillance by the requirement that producers can prove the contents of benzene in their finished products.

The monitoring of the damage or effects of exposure to benzene is regulated by NR 7 and 15 of the Ordinance / No MTb 3.214/1978, and the Normative / MTb No. 2 / 1995, which provide for the health surveillance of workers in the prevention of occupational exposure to benzene. According to this statement, are tools for health surveillance:

- clinical and occupational history and physical examination;
- CBC with platelet count and reticulocyte (monthly);
- epidemiological and toxicological risk groups, obtained by the evaluation of biological indicators of exposure. One of the biological indicators of exposure is the recommended concentration trans-trans muconic acid in urine, which VR is 0, 5 mg / g creatinine, with IBMP value of 1, 4

mg / g creatinine. The determination of the concentration of acid S-phenyl-mercapturicurine at the end of the working day is recommended by ACGIH (1998). Your IBMP is 25 g / g creatinine.

For pension purposes, should be consulted Service Order / Social Security No. 607/1998 approving technical standard on occupational intoxication benzene.

Federal Law No. 7.802/1989 and some state and local laws prohibit the use of organochlorine pesticides, including lindane, DDT and BHC and should not therefore be allowed its manufacturing and marketing. Workers exposed to pesticides should be monitored for effects arising from previous exposure. Epidemiological studies may be conducted to assess these effects.

It is recommended to check the adequacy of and compliance by the employer, the PPRA (NR 9), the PCMSO (NR 7) and other regulations -

health and environmental - in the states and municipalities. The NR 15 defines the LT concentrations in ambient air of various chemicals, for journeys of 48 hours a week.

The periodic medical examination aimed at identifying signs and symptoms for early detection of disease.

In addition to thorough clinical examination, we recommend the use of standardized instruments and laboratory testing appropriate to the risk factor identified. For exposure to arsenic, VR is up to 10 g / gcreatinine and the IBMP is 50 g / gcreatinine.

The procedures for health surveillance of workers exposed to ionizing radiation are

described in the protocol Malignant neoplasm of bone and articular cartilage of limbs, in Chapter 7

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the information systems of the SUS, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

PURPLE AND OTHER EVENTS HAEMORRHAGIC ICD-10

D69. -

1. DEFINITION OF THE DISEASE - DESCRIPTION

The spots on the skin caused by bleeding are called purple. Under the extension, may be called petechiae, when stippling, suffusion, when they are up to about 3 cm in size, or bruises, while more extensive. Other haemorrhagic manifestations are hematoma and bleeding of mucous (Epistaxis, hemoptysis, hematemesis, melena, rectal bleeding, hematuria, metrorrhagia) or serous (Hemothorax, hemopericardium, hemoperitoneum) or joints (hemarthrosis).

Bleeding may result from a number of abnormalities of hemostasis system, ie, blood vessels, platelets, coagulation and fibrinolysis.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Thrombocytopenia can be caused by:

- failure or decreased production of platelets, as in leukemia, lymphoma, aplastic anemia, paroxysmal nocturnal hemoglobinuria, thrombocytopenia induced by alcohol and other toxic thrombocytopenia in megaloblastic anemia in HIV infection, in myelodysplastic syndromes and in some cases of idiopathic thrombocytopenic purpura;
- platelet sequestration and hypersplenism;
- Accelerated removal of platelets, as in idiopathic thrombocytopenic purpura, systemic lupus erythematosus, in post-transfusion purpura, the thrombocytopenia associated with HIV infection in intravascular coagulation in thrombotic thrombocytopenic purpura;
- dilution in massive transfusions.

The thrombocytopenia induced by toxic drugs has been reported with the use of dozens of drugs, including NSAIDs (mefenamic acid, phenylbutazone, piroxicam, indomethacin, naproxen, aspirin, diclofenac, ibuprofen, etc.). Antibiotics β -lactam antibiotics, cardiovascular drugs, anticoagulants and thrombolytic agents, psychotropic drugs, narcotics and anesthetics, chemotherapeutic agents, antihistamines, radiological contrast, vitamins (C and E) and other drugs. Some foods and spices such as ginger, clove, onion, cumin

and garlic can also produce toxic thrombocytopenia.

The thrombocytopenia toxic nature of occupational exposure has been described in the benzene and ionizing radiation, due to thrombopoiesis ineffective with consequent thrombocytopenia, generally associated with cytopenia erythrocytic and granulocytic series (see aplastic anemia).

Other chemical agents that can cause occupational origin thrombocytopenia toxic by the same mechanism of action, are DDT, Lindane (hexachlorocyclohexane), the Letan, the 2, 2-dimethyl-phosphate dichlorovinyl and the organic arsenic. Chemical agents such as toluene-diisocyanate (TDI), the turpentine and the vinyl chloride can cause thrombocytopenia by immune mechanism. The vinyl chloride may cause thrombocytopenia by splenic sequestration in cases that occur with hypersplenism.

In workers exposed to these agents, in which other causes of purple and thrombocytopenia non-occupational, were excluded, the disease can be classified as work-related, in Group I of the Classification of Schilling, in which work can be considered as the necessary cause.

CLINICAL AND DIAGNOSTIC

The clinical picture is characterized by hemorrhagic manifestations

alone or combined. Regarding quantitative platelet disorders, the counts in the range of 40,000 to 60.000/mm³ can lead to post-traumatic bleeding and in the range 20.000/mm³ bleeding can occur spontaneously.

Bleeding can be diagnosed based on clinical history and physical examination, but the exact characterization of the framework depends on laboratory tests.

The initial assessment must be made by performing tests or examinations to identify coagulation defects:

- vascular diseases, such as tourniquet and bleeding time;
- quantitative platelet changes (such as platelets);
- qualitative platelet alterations (such as thromboelastography);
- activated partial thromboplastin time (PTTA) - intrinsic system;
- Prothrombin time - extrinsic system.

The erythrocyte and WBC complete the evaluation of the involvement of erythrocytic and granulocytic series. Laboratory tests more sophisticated and expensive can be given in a later stage.

TREATMENT AND OTHER CONDUITS

Limitation of exposure to toxins and cited the use of drugs potentially causing thrombocytopenia or platelet dysfunction. In severe cases with hemorrhagic manifestations and is indicated for the transfusion of platelet concentrate.

PREVENTION

The prevention of purpura and other hemorrhagic manifestations related to work based on monitoring of environments, working conditions, and effects or health hazards, as described in the introduction to this chapter. Environmental control of exposure to benzene, ionizing radiation, to vinyl chloride and the other substances mentioned in item 2 can reduce the incidence of disease in occupational groups at risk.

The procedures for health surveillance of workers exposed to benzene and ionizing radiation are described, respectively, in protocols Aplastic anemia due to other external agents, this chapter, and Malignant neoplasm of bone and articular cartilage of the members in the chapter for the vinyl chloride, consult

Protocol Angiosarcoma of the liver.

Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;

- notify the case to the information systems of the SUS, the DRT / MTE and the labor union;

- provide for the issuance of CAT, where the worker is insured by the SAT for Social Security

- direct the employer to adopt the technical and managerial procedures to eliminate or control of risk factors

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[229-251]

Agranulocytosis (Neutropenia Toxic) ICD-10 D70

1. DEFINITION OF THE DISEASE - DESCRIPTION

Agranulocytosis is the term used to define the reduced number of granulocytes (neutrophils, eosinophils and basophils) in peripheral blood as a result of a disturbance in the bone marrow, usually by myelosuppressive effect of toxic chemicals.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The main causes of neutropenia can be classified as:

Abnormalities of bone marrow

INJURY OF THE BONE MARROW

- Chemical Agents: benzene, dinitrophenol, pentachlorophenol, nitrous oxide, 2-ethoxyethanol (cellosolve), arsenic, Lindane (HCH or BHC);

- ionizing radiation;

- drugs, cytotoxic agents and non-cytotoxic;

- certain congenital neutropenia and hereditary;

- immune-mediated conditions;

- infections such as hepatitis, parvovirus, HIV, M. tuberculosis, M. kansasii;

- replacement of bone marrow: leukemias, lymphomas and other malignancies.

DEFECTS OF MATURATION

- acquired: deficiencies Folic Acid and vitamin B12;
- clonal neoplasms and other diseases;
- congenital neutropenia;
- myelodysplastic syndromes;
- acute non-lymphocytic leukemia;
- noturnal paroxysmal hemoglobinuria.

Abnormalities in the peripheral blood compartment

DIVERSION OF NEUTROPHILS STOCK TANK FOR MARGINAL

- pseudoneutropenia benign hereditary;
- acquired: acute (severe bacterial infection associated with endotoxemia);
- chronic (protein-calorie malnutrition, malaria).

KIDNAPPING

- lung: leucoaglutinação mediated by complement;
- spleen: hypersplenism.

Abnormalities in the extravascular compartment

INCREASE IN USE

- severe infection bacterial, fungal, viral or rickettsial;
- anaphylaxis.

DESTRUCTION

- antibody-mediated rheumatic diseases and drugs;
- hypersplenism.

Among the occupational agents stand out from the benzene and ionizing radiation, whose mechanism of action is the same as described for other purposes haematotoxic. Also important are phenol

(Dinitrophenol pentachlorophenol), the arsenic, the nitrous oxide and the hydroxibenzonitrite.

In workers exposed to these agents, in which other causes of agranulocytosis or neutropenia toxic non-occupational were excluded, they can be classified as work-related diseases, the Group I of the Classification of Schilling, in which work can be considered as the necessary cause.

3. CLINICAL AND DIAGNOSTIC

Neutrophils are granulocytes present in greater quantities in the blood, so the neutropenia laboratory finding is the most obvious and major clinical importance. In agranulocytosis fulminant, symptoms

appear quickly. Tremors, high fever and exhaustion are evident. Gangrenous ulcers can be detected in the gums, tonsils, soft palate, lips, tongue or pharynx and, less commonly, skin, nose, vagina, uterus, rectum or anus. There may be regional adenopathy. Death occurs quickly, usually three to nine days.

In other patients, the course is insidious and the clinical manifestations of infectious processes are as a result of increased susceptibility to infections.

In agranulocytosis isolated, deficiency of granulocytes stands in the count, but other types of leukocytes may also be reduced numerically.

In acute cases, the total leukocyte count, usually is less than 2.000/mm³, often below 1.000/mm³. The granulocytes may be completely absent and lymphocytes and monocytes can increase in relative and absolute terms.

In chronic cases, the neutropenia can be a slow rise and leukocyte can not fall below 2.000/mm³. In these cases, granulocytopenia may be less pronounced.

When the agranulocytosis is isolated marrow examination may show the erythrocytic and megakaryocytic series were normal. The most remarkable aspect is the absence of granulocytes (polymorphonuclear cell nuclear metamyelocytes and myelocytes).

Criteria for staging of disorders caused by deficiency of white blood cells, according to the AMA, are presented in Table XVII.

4. TREATMENT AND OTHER CONDUITS

The most important step is identifying the offending agent and the possible cessation of exposure or use, in case medications. In fulminant cases, the use of empirical antibiotics after samples for culture of achievement, it is mandatory. Oral hygiene and general care deserve attention. The stimulation of granulocyte production by using granulokinases (G-CSF and GM-CSF) appears to be a promising therapy. Transfusion of neutrophils is complicated by the frailty and brief survival of these cells, among other factors.

5. PREVENTION

The prevention of agranulocytosis related to work based on monitoring of environments, working conditions and the effects or health hazards, as described in the introduction to this chapter. Environmental control of exposure to benzene, ionizing radiation, derived from phenol, arsenic, nitrous oxide and hidroxibenzonitrito, among other substances, may reduce the incidence of disease in occupational groups at risk.

The procedures for health surveillance of workers exposed to benzene and ionizing radiation are described in the protocols Plastic anemia due to other external agents, this chapter, and malignant neoplasm of bone and articular cartilage of limbs,

Thedinitrophenol and pentachlorophenol are used as fungicides, having established his control by Federal Law No. 7.802/1989. Some state and local laws prohibiting their use as wood preservatives and natural fibers, indicating the need for their replacement by less tóxicos.Recomenda observe compliance by the employer, the NRR, Ordinance / No MTb 3.067/1988, especially NRR 5, which deals with chemicals (pesticides and the like), fertilizers. Special attention should be given the protection of workers involved in the activities of preparation of grout and application of these products.

It is recommended to check the adequacy of and compliance by the employer, the PPRA (NR 9), the PCMSO (NR 7) and other regulations - health and environmental - in the states and municipalities. The NR 15 d defines the LT concentrations in ambient air of various chemicals, for journeys of 48 hours a week.

The periodic medical examination should be geared towards identifying signs and symptoms that facilitate early detection of disease. Besides a thorough clinical examination, we recommend the use of standardized instruments and laboratory testing appropriate to the risk factor identified. Exposed to arsenic in: concentration of arsenic in urine - VR up to 10 mg / g creatinine IBMP and 50 mg / g creatinine.

In exposed to pentachlorophenol: dose in urine - VR 2 mg / g creatinine.

Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the information systems of the SUS, the DRT / MTE and the labor union
- provide for the issuance of CAT, where the worker is insured by the SAT for Social Security as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

OTHER SPECIFIED DISORDERS OF WHITE BLOOD CELLS:
Leukocytosis, leukemoid reaction ICD-10 D72. 8

1. DEFINITION OF THE DISEASE - DESCRIPTION

Leukocytosis refers to an increased number of leukocytes in peripheral blood, exceeding 10.000/mm³.

Left shift is the emergence of more than 600 rods / mm³ in the peripheral blood or at least a metamyelocytes in the blood stream.

A leukemoid reaction is the appearance of at least one myelocytes in the blood stream, featuring a left shift more intense diversion hierarchy, with a predominance of mature cells, without gaps, without the presence of blasts. In general, platelets and red blood cells not are affected.

These terms have less clinical significance compared to those that identify the type of leukocytes, predominantly increased. The terms neutrophilia, eosinophilia, basophilia, lymphocytosis and monocytois suggest specific diagnostic considerations.

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Leukocytosis, mainly neutrophilia is a common finding that occurs in response to acute and subacute inflammatory processes, such as infections, bacterial infections, rheumatic and autoimmune disorders, trauma and hemorrhage, tumors, and other causes. Work in pathology, the increased number of leukocytes may be an early sign of leukaemogenic effect of exposure to benzene and ionizing radiation.

Paradoxically, this effect can also hematological precede the onset of aplastic anemia.

In exposed workers, in which other causes of leukocytosis and leukemoid reaction non-occupational were excluded, they can be classified as work-related diseases, the Group I of the Classification of Schilling, in which occupational exposure to benzene and ionizing radiation can be considered a necessary cause.

3. CLINICAL AND DIAGNOSTIC

There are no specific symptoms of these hematological changes in cases secondary to exposure to benzene and ionizing radiation. Cases secondary to inflammatory processes specific symptoms of the pathology dominates the picture. The absence of splenomegaly, the rate of alkaline phosphatase and increased picture resolution, linked temporally with the decline of the underlying disease, are some of the findings leukemoid reaction neutrophilia that help differentiate it from chronic myelogenous leukemia.

4. Treatment AND OTHER CONDUITS

This is not the changes in blood but the underlying pathology. Cases secondary to exposure to benzene and ionizing radiation, it is important to stop the exposure and maintain surveillance of the worker.

5. PREVENTION

The prevention of other specified disorders of blood cells related to the white paper is based surveillance environments, working conditions and the effects or health hazards, as described in the introduction to this chapter. Environmental control of exposure to benzene and ionizing radiation reduces the incidence of disease in occupational groups at risk.

The procedures for health surveillance of workers exposed to benzene and ionizing radiation are described, respectively, in protocols Aplastic anemia due to other external agents (8.3.4) this chapter, and Malignant neoplasm of bone and articular cartilage of the members (7.6.7)

Suspected or confirmed the relationship of disease with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the information systems of the SUS, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

Methemoglobinemia ICD-10 D74. -

1. DEFINITION OF THE DISEASE - DESCRIPTION

Methemoglobinemia is present in concentrations higher than normal (1%) of methemoglobin in the blood. Methemoglobin is formed by oxidation of the atom iron the ferrous form (Fe^{2+}) to form ferric (Fe^{3+}), making the molecule unable to bind to oxygen-methemoglobinemia may be inherited or acquired secondary to exposure to toxins and the use of drugs

2. EPIDEMIOLOGY - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Among the drugs involved in methemoglobinemia toxic, are:

- amyl nitrite; •dapsone;
- sodium nitrite; •benzocaine;
- silver nitrate; •prilocaine;

- nitroglycerin; •primaquine;
- quinones; •resorcinol;
- sulfonamides; •phenazopyridine.

The methemoglobinemia hereditary diseases are rare.

Among the chemical nature of producing occupational methaemoglobinemia are highlighted the aromatic amines and its derivatives. The aromatic amines are chemicals derived from aromatic hydrocarbons (Benzene, toluene, naphthalene, anthracene, etc.). By substitution of at least one atomhydrogen by a group amino (-NH₂), exemplified by the following substances:

- aniline; •acetanilina;
- dimethylaniline; •benzidine;
- dietilanilina; •o-toluidine;
- chloroaniline; •o-dianisidine;
- nitroaniline; •3, 3-dichlorobenzidine;
- nitrobenzene; •4-aminodifenilo;
- toluidine; •naphthylamine;
- clorotoluidina; •Aminoanthracene.
- phenylenediamine;

In exposed workers, in which other non-occupational causes of methemoglobinemia were excluded, it can be classified as work related disease, the Group I of the Classification of Schilling, where occupational exposure to aromatic amines can be regarded as a necessary cause.

3. CLINICAL AND DIAGNOSTIC

Symptoms vary in intensity, but often are mild. The concentrations of 10 to 25% methemoglobin produce cyanosis, but they are tolerated without apparent ill effect, 35 to 40% can be felt a slight dyspnea on exercise, headache, fatigue, palpitations and dizziness. Lethargy and stupor can occur with concentrations of 60%. The lethal concentration for adults is probably higher than 70%.

Cyanosis without hypoxemia should suggest the possibility of methemoglobinemia. Peripheral blood is maroon. The diagnosis is confirmed by the determination of methemoglobin blood.

The differential diagnosis is done with the Sulfhemoglobinemia and differentiation can be made by adding a few drops of potassium cyanide 10% blood, resulting in rapid production of cyanmethemoglobin, which has bright red color, which does not occur with Sulfhemoglobinemia.

4. TREATMENT AND OTHER CONDUITS

In the absence of symptoms, cessation of exposure to an offending agent is sufficient to permit the conversion of methemoglobin in hemoglobin through physiological mechanisms. In symptomatic cases, it is indicated the use of methylene blue intravenously. The initial dose is 1 mg / kg (1% solution) in five minutes. A second dose of 2 mg / kg may be given if cyanosis does not clear up within an hour.

5. PREVENTION

The prevention of Acquired methemoglobinemia related to work based on monitoring of environments, working conditions and the effects or health hazards, as described in the introduction to this chapter.

The environmental control of industrial processes that generate aromatic amines can effectively reduce the incidence of disease in occupational groups at risk.

The environmental control measures aimed at the elimination or reduction of exposure to concentrations close to zero, by:

- entrapment and isolation procedures of work sectors;
- use of hermetically sealed systems in the industry;
- hygiene standards and strict security, with adoption of exhaust ventilation systems adequate and efficient;
- systematic monitoring of concentrations of agents in ambient air;
- changes in work organization that allow reducing the number of exposed workers and exposure time;
- measures of general cleaning of work environments and facilities for personal hygiene, as resources for bathing, washing hands, arms, face and exchange of clothing;
- provision by the employer of appropriate personal protective equipment in good repair, as indicated in a complementary way to measures of collective protection.

The respiratory protective masks should be used as a temporary measure in emergencies. When the measures of collective protection is inadequate, these should be carefully indicated for some sectors or functions. Workers must be trained appropriately for their use. Masks should be of quality and relevance to exposures with chemical or dust filters, specific for each substance handled or groups of substances which may be retained by the same filter. Filters should be changed strictly according to the manufacturer's recommendations. The Normative / MTb No. 1 / 1994 establishes the technical regulation on the use of respiratory protection equipment.

It is recommended to check the adequacy of and compliance by the employer, the PPRA (NR 9), the PCMSO (NR 7) and other regulations -

health and environmental - in the states and municipalities. The NR 15 defines the LT concentrations in ambient air of various chemicals, for journeys of 48 hours a week, for example:

- aniline: 4 ppm or 15 mg/m³;
- diethylamine: 20 ppm or 59 mg/m³;
- hydrazine / diamine: 0, 08 or 0 ppm, 08 mg/m³.

The periodic medical examination aimed at identifying signs and symptoms for early detection of disease.

In addition to thorough clinical examination, we recommend the use of standardized instruments and laboratory testing appropriate to the risk factor identified. In the case of occupational exposure to agents metahemoglobinizantes, should be conducted biological monitoring of occupational exposure through the determination of methemoglobin in the blood, as determined by NR 7 (PCMSO), the Ministry of Labour. The biological indicators of exposure to aniline are the concentration of p-Aminophenol in urine, which IBMP is 50 mg / g creatinine, and determination of methemoglobin in the blood, where VR is up 2% and IBMP is 5%.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- guidance to the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

CHAPTER 15

DISEASES ENDOCRINE, NUTRITIONAL AND METABOLIC WORK-RELATED

(Group IV ICD-10)

9.1 INTRODUCTION

The effects or damage to the endocrine, nutritional and metabolic status, resulting from environmental or occupational exposure to toxic substances and agents are still poorly understood. However, although requiring further study, the following work situations are recognized as capable of producing disease:

- use of vibrating tools such as hammers tires. Associated with Raynaudg syndrome, a peripheral vascular disease (see protocol in Chapter 14) has been observed impairment of the endocrine and central nervous expressed by dysfunction of autonomic brain centers that need to be better assessed;

- extraction and handling of pumice stone, causing adrenal failure;

- production and use of derivatives carbamic acid (Carbamates), used as pesticides, herbicides and nematocides. Thethiocarbamate are used also as vulcanization accelerators and their derivatives used in the treatment of malignant tumors, hypoxia, neurological disorders and diseases caused by radiation. For endocrine mechanism, are mutagenic and embryotoxic;

- in exposed Lead has been observed a strong inverse correlation between blood lead and vitamin D levels, altering the homeostasis of intracellular and extra calcium and interfering with the growth and maturation of teeth and bones. It has also been reported the occurrence of hypothyroidism due to an impairment of the pituitary;

- exposure carbon disulfide (CS₂) is recognized for its effects on lipid metabolism, accelerating the process of atherosclerosis (also known as arteriosclerosis).

The scientific literature has given prominence to the role played by certain synthetic chemicals, endocrine disrupters, which interfere with natural hormones, neurotransmitters and growth factors to produce disease often difficult to recognize. For example, intrauterine exposure to diethylstilbestrol (DES), a synthetic hormone, may lead to changes in the reproductive system of women, as the cornification of the vaginal epithelium, vaginal clear cell adenocarcinoma and other problems for reproduction, which will only be identified in adulthood.

Other effects of endocrine disruptors, including the reduction of intelligence quotient (IQ), behavioral changes and immune, thyroid disease and reproductive tract changes, such as hypospadias, cryptorchidism, testicular cancer, semen quality and sperm count, may remain undiagnosed and / or unconnected to the previous exposure, over the life of affected individuals.

A large number of substances have been recognized as endocrine disrupters and toxic to reproduction, particularly pesticides, herbicides, fungicides, insecticides, industrial chemicals and nematocides as OH 4-alkylphenol, 4-OH biphenyl, cadmium, dioxin, lead, mercury, PBB, PCB, PCP, phthalates, styrene, among others. They act by different mechanisms of action are grouped into six general categories:

- binding to receptors and enhancing the effects as antagonists;
- blocking the receptors and inhibiting effects as antagonists;
- working directly with the endogenous hormones;
- indirectly interfering in endogenous hormones or other natural chemical messengers;
- altering steroidogenesis, metabolism and excretion;
- altering levels of hormone receptors.

It is important to remember that a contaminant may interfere with the homeostasis of more than one way and that in some cases, the toxicity depends greatly on the time of exposure than the dose.

The effects of endocrine disruptors during the development mean a challenge for professionals on the insidious character and often act more in the reduction of functions than causing a disease in itself. Represents a new frontier of knowledge to which professionals in the Occupational Health should contribute.

The prevention of endocrine, nutritional and metabolic diseases related to work based on the surveillance procedures to the health, the environment and working conditions.

It is based on medical knowledge, clinical, epidemiological, occupational hygiene, toxicology, ergonomics, psychology, among other disciplines in the perception of employees on work and health and technical standards and regulations in place, involving:]

- prior knowledge of the activities and work places where there are chemicals or physical agents or biological risk factors arising from work organization, potentially causing disease;

- identifying problems or potential harm to health arising from exposure to identified risk factors;

- identifying and proposing measures to control that must be taken to eliminate or control exposure to risk factors and for protection of workers;

- education and information to workers and employers.

Upon confirmation of the diagnosis and establish its relationship to work, following the procedures described in Chapter 2, the health services responsible for health care worker should implement the following actions:

- assessing the need for removal (temporary or permanent) worker exposure, sector of employment or work as a whole;

- whether the worker is insured by the Social Security SAT, request the issuance of CAT to the company, fill in the LEM and forward to the INSS. In case of refusal to issue the CAT by the company, the doctor (or medical services) should do so;

- monitoring of progress, and worsening record of clinical worsening and their relationship to return to work;

- notification of this disease to the information system of morbidity of the SUS, the DRT / MTE and the union of the employee;

- implement the epidemiological surveillance activities aimed at the identification of other cases, through active search in the same company or workplace or other companies of same industry in the geographic area;

- if necessary, supplement the identification of the agent (chemical, physical or biological) and working conditions determine the disorder and other risk factors that may be contributing to the occurrence;

- inspection at the company or work environment where the patient or worked in other companies of same industry in the geographic area, identifying risk factors for health and collective protection measures and personal protective equipment used;

- recommendation to the employer on the measures of protection and control to be adopted, informing the workers.

The measures of protection and prevention of exposure to risk factors at work include:

- replacement of production technologies by less hazardous to health;

- isolation of the agent / substance or process enclosure, preventing exposure;

- adoption of local exhaust ventilation systems and general adequate and efficient;

- use of exhaust fume hoods;
- control spills and incidents through preventive and corrective maintenance of machinery and equipment and monitoring compliance;
- setting standards of hygiene and safety, for example, systematic environmental monitoring;
- adoption of safe systems of work, operational and transportation;
- classification and labeling of chemical substances toxicological properties and toxicity;
- information and risk communication to workers;
- maintenance of proper conditions and general environment of comfort for employees and facilities for personal hygiene and adequate sanitary facilities, restrooms, showers, sinks with clean running water and an abundance of clothing and cleaned daily;
- decrease the exposure time and the number of exposed workers;
- supply of personal protective equipment, proper maintenance and indicated, in complement to measures of collective protection.

LIST OF DISEASES ENDOCRINE, NUTRITIONAL AND METABOLIC-RELATED WORK IN ACCORDANCE WITH THE ORDINANCE / MS No. 1.339/1999

•Hypothyroidism due to exogenous substances (E03. -) •Other porphyria (E80.2)

HYPOTHYROIDISM DUE TO EXOGENOUS SUBSTANCES ICD-10 1 DEFINITION OF DISEASE - DESCRIPTION

Hypothyroidism is the clinical syndrome caused by a reduction in the circulation of thyroid hormone (TH) or, more rarely, peripheral resistance to its action. Consequently, there is general decrease in metabolic processes with deposition of glucosamine in intracellular spaces, especially in the skin and muscles, but affecting all organs and systems. Because these substances are hydrophilic mucopolysaccharides, provide more intense edema in the skin, subcutaneous tissue, skeletal muscles and heart. The symptoms in its most advanced form is myxedema. May be reversible in adults with treatment. However, the somatic and mental retardation may be associated with permanent disability when the HT in the pre-natal and newborn, leading to cretinism, a severe and irreversible effect of maternal hypothyroidism.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The primary form of hypothyroidism corresponds to more than 90% of cases. The association with work has been described in workers exposed to:

- Lead or its toxic compounds;
- halogenated hydrocarbons (chlorobenzene and their derivatives);
- thiouracils;
- tiocinatos;
- thiourea.

The diagnosis of hypothyroidism in workers exposed to these chemicals, excluded other non-occupational causes, provides the basis for the disease in Group I of the Classification of Schilling in that work plays the role of necessary cause.

3 CLINICAL AND DIAGNOSTIC

Clinical manifestations of hypothyroidism are varied. Depends on the cause, duration, severity and stage of life when TH deficiency occurs. The disease is insidious and appearance may vary from normal to the myxedema.

Appear dismal, cold intolerance, hoarse voice, slurred speech, dry skin, scaly and infiltrated (myxedema), eyelid edema, hair and nails dry and brittle, pale. In more advanced forms, the skin has a waxy appearance, there is reduced intellectual ability, apathy and drowsiness. The heart rate is reduced, there is cardiomegaly and pericardial effusion. It is common to evolve, still to hypermenorrhea, anovulation, decreased libido, impotence, and coma. In the congenital form, there is mental retardation, which is aggravated by the delay in the initiation of therapy.

Myxedema coma is rare and severe. Most cases are precipitated by acute infection, tranquilizers and sedatives, painkillers or anesthetics. The body temperature becomes very low, the skin is cold and dry, the repercussions extend osteotendinosos. Surge sphincter incontinence, hypotension and coma. The mortality rate reaches 60% of cases.

The laboratory diagnosis is confirmed. The doses of T3 and T4 are low. The TSH is elevated in hypothyroidism primary, normal or decreased in the pituitary or hypothalamic forms. There is also hypoglycemia, hyponatremia, and elevation of pO₂ acidemia. The ECG revealed bradycardia, low voltage and nonspecific T wave. Hypercholesterolemia is finding ever-present. Asthenic patients with chronic diseases may show clinical examination consistent with hypothyroidism, with no change in thyroid function.

4 TREATMENT AND OTHER CONDUITS

It is based on hormone replacement. It is used to L-thyroxine (T4) at doses of 2, 25 mg / kg / day for adults.

In young people, starting with 100 mg / day and increase it if necessary, at monthly intervals until the appropriate dose.

In patients aged beyond 45 years or hypothyroidism Long-term, but without heart disease, the initial dose should be 50 mg / day with dose increases up to 50mg every month until the desired setting. In the elderly or when there is coronary artery disease, the initial dose should be 25mg / day with increases of 25g per month, not to exceed 100mg / day. The cardiological control must be strict.

Replacement in excess TH can cause osteoporosis and cardiovascular overload and, if insufficient, does not restore metabolic balance. It is therefore necessary to control clinical and laboratory treatment. The most sensitive indicator for assessment is the TSH.

In myxedema coma should be used in the T4 dose from 200 to 300 g by nasogastric tube on the first day followed by 100g on day 50 andg from the third day of treatment. It should also be used T3, 25g of 12/12 hours to improve the level of consciousness. Hydrocortisone should be used at a dose of 100 mg intravenously, immediately and up to 6 / 6 hours. It should provide proper heating for the patient, ventilatory support, adequate hydration and correction of hydro-electrolyte disturbance and treat the causes triggering.

5 PREVENTION

The surveillance hypothyroidism due to exogenous substances follow the procedures outlined in the introduction to this chapter for the elimination or control of occupational exposure to Lead and its toxic compounds; halogenated hydrocarbons (chlorobenzene and their derivatives); thiouracils; thiocyanate and thiourea; and measures to promote the health of exposed workers. The environmental control measures aimed at the elimination or reduction of concentration levels of these substances, through:

- entrapment and isolation procedures of work sectors;
- use of hermetically sealed systems in the industry;
- hygiene standards and strict safety, exhaust ventilation systems adequate and efficient;
- monitoring of concentrations of smoke, haze and dust in the air;
- changes in work organization that allow reducing the number of exposed workers and exposure time;
- general cleaning arrangements of the work environment, facilities

for personal hygiene, resources for bathing, washing hands, arms, face and exchange of clothing;

- provision by the employer of appropriate personal protective equipment in good repair, as indicated in a complementary way to measures of collective protection. It is recommended to check the suitability and adoption by the employer of the control measures of occupational risk factors and health promotion identified in the PPRA (NR 9) and PCMSO (NR 7), the Ordinance /

MTb No. 3.214/1978, and other regulations - health and environmental - in the states and municipalities.

The LT 15 for NR defined by exposure to substances potentially capable of causing hypothyroidism are:

- Lead: 0, 1 mg/m³;
- chlorobenzene: 59 ppm or 275 mg/m³.

These LT can not be related to the endocrine effects, since these are still largely unknown.

Thus, it is recommended to watch shows with more indexes low.

Some pesticides, especially fungicides of the group of dithiocarbamates, which contain as an impurity etilenoetiluréia

(ETU) or manganese, herbicides and derivatives phenoxyacetic acid can cause damage to the endocrine system and teratogenesis. Its manufacturing and marketing are banned in some countries. In Brazil, these and other pesticides have their production, marketing, use, transport and disposal as defined by Federal Law No. 7.802/1989. Some states and municipalities have regulations still to be obeyed. Observe the NRR, the Ordinance / No MTb 3.067/1988, especially NRR 5, which deals with chemicals (pesticides and the like), fertilizers. Special attention should be given the protection of workers involved in the activities of preparation of grout and use of these products.

The periodic medical examination aimed at identifying signs and symptoms for early detection of disease, through:

- clinical evaluation using a standardized protocol and careful physical examination;
- exams guided by occupational exposure;
- epidemiological information.

For workers exposed to lead, the main biological indicators of exposure are:

- Blood lead concentration (PbB) - VR up to 40 g/100 ml of IBMP and 60 mg / 100 ml. (A Serum lead levels reflects the absorption of the metal in the weeks preceding the sample collection or deposit mobilization bone);

- amino acid concentration delta Levulinic in urine (ALA-U) - VR up to 4, 5 mg / g creatinine IBMP and up to 10 mg / g creatinine;
- zincoprotoporfirina concentration in blood (ZPP) - VR up to 40 g/100 ml and 100 g/100 ml of IBMP.

The dosage of Lead serum reflects the absorption of the metal in the weeks preceding sampling of amostraou the mobilization of bone deposits.

Workers exposed to pesticides should be monitored for effects arising from exposure

stunted and present, through research of signs and symptoms and conducting periodic complete blood counts and other tests available, depending on the specific product.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
 - examine the exposed, to identify other cases;
 - notify the case to the information systems of the SUS, the DRT / MTE and the labor union;
 - provide for the issuance of CAT, where the worker is insured by the SAT for Social Security
- ;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

The procedures for health surveillance of workers exposed to Lead are described in the protocol Lead colic (16.3.6)

”
Other porphyrias ICD-10 E80.2

1 DEFINITION OF DISEASE - DESCRIPTION

Porphyrias disorders are caused by partial deficiencies of one of the eight enzymes involved in heme production. A separate form is associated with a deficiency in six of the eight enzymes referidas.Characterizam up by the formation and excessive excretion of porphyrins or their precursors, intermediates of heme synthesis.

They appear almost always inborn errors of metabolism, except in cases of porphyria cutanea tarda, that can be inherited or acquired, and porphyrias secondary to other conditions or induced by certain chemicals.

The porphyrias are rare, as manifested by disturbances neuroviscerais and / or skin photosensitivity.

They may present as acute intermittent porphyria (PIA) Porphyria cutanea tarda (PCT) and erythropoietic protoporphyria (PE).

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Situations of occupational and environmental exposure to organochlorines has been classically associated with porphyria cutanea tarda. The best known episode is the outbreak in Turkey (hence the name Porphyria turcica) in the 50's, caused by accidental ingestion of massive hexachlorobenzene (HCB). Other episodes have

been reported, associated with the production of chlorinated solvents (Perchloroethylene), production and use of polychlorinated biphenyls (PCB) of Pentachlorophenol exposure to 2, 4-dichlorophenol (2, 4-D) and at 2, 4, 5-Trichlorophenol (2, 4, 5-T). Other agents are related monochlorobenzene and the monobromobenzene. A number of substances used in medical therapy may precipitate porphyria, among them valproic acid, barbiturates, carbamazepine, chloramphenicol, oral contraceptives, chlorpropamide, danazol, dapsone, phenytoin, ergot preparations, ethanol, glutethimide, griseofulvin, imipramine, meprobamate, methyl dopa, progestins and sulfonamides. Attacks can also be precipitated by low-calorie diet, stress, infections and other diseases with metabolic requirements or excessive surgery. On diagnosis of porphyria acquired by the exposed workers, other causes must be framed in Group I of the Classification of Schilling, where work is defined as the necessary cause.

3 CLINICAL AND DIAGNOSTIC

The Porphyria cutanea tarda (PCT) is the most common form of porphyria. The enzyme is affected uroporphyrinogen (urogenio)-decarboxylase, which is in step 5 of heme biosynthesis. The major porphyrins to accumulate in the plasma are uroporphyrin and 7-carboxylate-porphyrin. The urogenio and 7-carboxylate-porphyrin are responsible for the photosensitivity observed in the PCT.

It is characterized clinically by cutaneous photosensitivity, blistering and scarring, pigmentation and hypertrichosis. There is no acute episodes. The onset of manifestations is insidious and patients do not involve the skin lesions to sun exposure. Injuries trivial in the back of hands, arms, feet or faces leads to the formation of vesicles that rupture, forming an open wound and later scarring and fibrosis. Can be observed hyperpigmentation and hyperkeratosis. The urine is orange-red or brown.

The incidence of hepatocellular carcinoma seems to increase.

The diagnosis of porphyrias based on clinical and laboratory manifestations and history of exposure to toxic substances, for example, hexachlorobenzene.

The exams can help diagnose:

- determination of uroporphyrin in urine: high (above 800 µg/24h) and faeces: normal;

- Liver biopsy revealed subacute hepatitis or cirrhosis. The red fluorescence in ultraviolet light can demonstrate uroporphyrin.

4 TREATMENT AND OTHER CONDUITS

Avoid contact with or use drugs that can precipitate porphyria, through the identification of substances and situations of risk to which the patient is exposed. We recommend the use of sunscreen (protection factor

Solar/FPS-26 or higher) and avoid the sun. The use of β-carotenemay offer some protection to the effects of lightning. Sun. The chlorpromazine can be safely used to treat pain and changes in behavior. Analgesics and opioids are also safe and effective during crises. The food supply of carbohydrates must be at least 300 g / day.

Venous infusions hemin (Derived from processed red blood cell heme) inhibits ALA-synthase and reduce the production of ALA and PBG, but have modest efficacy. The use of β-blockersis effective in controlling hypertension and tachycardia. Phlebotomy for treatment of hemochromatosis Surte benefit against porphyria cutanea tarda.

5 PREVENTION

Prevention of cases of porphyrias other work-related based on monitoring of environments, working conditions and the effects or health hazards,as described in the introduction to this chapter.

Environmental control of occupational exposure to chlorophenol in the production or application of 2, 4-dichlorophenol (2, 4-D) and 2, 4, 5 -

Trichlorophenol (2, 4, 5-T), the production of chlorinated solvents (Perchloroethylene), production and use of polychlorinated biphenyls (PCBs) and Pentachlorophenol may reduce the incidence of disease in occupational groups at risk. AMESD environmental control aimed at the elimination or reduction of exposure through:

- entrapment and isolation procedures of work sectors;

- use of hermetically sealed systems in the industry;
- hygiene standards and strict security, with adoption of exhaust ventilation systems adequate and efficient;
- systematic monitoring of concentrations of agents in ambient air;
- changes in work organization that allow reducing the number of exposed workers and exposure time;
- measures of general cleaning of work environments and facilities for personal hygiene, as resources for bathing, washing hands, arms, face and exchange of clothing;
- provision by the employer of appropriate personal protective equipment in good repair, as indicated in a complementary way to measures of collective protection.

It is recommended to check the suitability and adoption by the employer of the control measures of occupational risk factors and health promotion identified in the PPRA (NR 9) and PCMSO (NR 7), the Ordinance / MTb No. 3214 / 78

and other regulations - health and environmental - in the states and municipalities

.

The LT defined by the NR 15 to 48 hours of weekly exposures to substances that can potentially cause porphyrias are:

- Perchloroethylene: 78 ppm or 525 mg/m³;
- chlorobenzene: 59 ppm or 275 mg/m³.

The observance of these LT can not be related to the endocrine effects, since they are still little

known. Thus, it is recommended to monitor exposures with lower limits.

The pentachlorophenol its use has been prohibited or restricted in some countries, however, Brazil is still used as a fungicide and wood preservative. The 2, 4-dichlorophenol (2, 4-D) and 2, 4, 5-Trichlorophenol (2, 4, 5-T) are banned in several countries, but are also approved and used in Brazil as herbicides. In Brazil, these and other pesticides have their production, marketing, use, transport and disposal as defined by Federal Law No. 7.802/1989. Some states and municipalities have regulations still to be obeyed.

The NRR, Ordinance / No MTb 3.067/1988, especially NRR 5, which deals with chemicals and pesticides, fertilizers and should be followed. Special attention should be given to the protection of workers

involved in the activities of preparation of grout and application of these products.

The periodic medical examination aimed at identifying signs and symptoms for early detection of disease.

Uses clinical and occupational history, physical examination and laboratory, epidemiological and toxicological evaluation by biological indicators, when available. The IBMP, to monitor the exposure Pentachlorophenol

is 2 g / gcreatinine (Urine).

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the information systems of the SUS, the DRT / MTE and the union.

CHAPTER 16

MENTAL DISORDERS AND BEHAVIOUR RELATED WORK

(Group V of ICD-10)

10.1 INTRODUCTION *

According to WHO estimates, the minor mental disorders affect about 30% of workers employed, and severe mental illness, about 50-10%. In Brazil, the INSS data on granting social benefits of sickness, incapacity for work exceeding 15 days and permanent disability, permanent disability for work, show that mental disorders, especially the chronic alcoholism, ranks third among the causes of these occurrences (Medina, 1986).

In our society, work is a mediator of social integration, both for its economic value (subsistence), due to the cultural (symbolic) and therefore of fundamental importance in subjectivity, in the way of life and, therefore, health physical and mental health of people. The contribution of labor to changes in the mental health of people takes place from a wide range of aspects: from off factors such as exposure to certain toxic agent, to the articulation of complex factors related to work organization, as the division and division of tasks, policies, people management and organizational hierarchical structure. The mental disorders

and behavior related to work result, therefore, not isolated factors, but the work contexts in interaction with the body and the psychic apparatus of the workers. The actions involved in the act of working can achieve the body of workers, producing biological damage and dysfunction, but also psychological reactions to work situations pathogenic, and may trigger psychopathological processes specifically related to conditions of work performed by the worker.

Due to the prominent place that work occupies in people's lives and a source of livelihood security and social position, lack of work or even the threat of job loss cause suffering psíquicoG by threatening the livelihoods and material life of worker and his family. At the same time undermines the subjective value that the person is assigned, generating feelings of capital loss, anguish, insecurity, hopelessness and despair, featuring paintings of anxiety and depression.

The current world economic scenario, in which the conditions of job insecurity, underemployment and the segmentation of the labor market are increasing, reflected in the internal processes of production restructuring, downsizing of staff, technological resources, impacting the

mental health workers.

The work covers also a key place in the dynamics of affective investment of pessoas. Condições favor of free use of the skills of workers and the control of labor by workers have been identified as key requirements for the job can provide pleasure, wellbeing and health, failing to cause disease. On the other hand, the work lacks meaning, without social support, unrecognized or that would constitute a source of threat to physical and / or psychological, can trigger psychological distress.

Various situations as a failure, an accident at work, a position change (rise or fall) in the hierarchy often determine various psychopathological, since the so-called adjustment disorders or reactions to estresse until severe and incapacitating depressions, varying according to characteristics of the context of the situation and the mood of the individual to respond to them.

The process of communication within the work environment, shaped by organizational culture is also an important factor in determining mental health. Environments that prevent spontaneous communication, expressions of dissatisfaction, employees' suggestions regarding the organization or the work performed will lead to tension and therefore pain and mental disorders. Often, the suffering and dissatisfaction of worker forward not only by the disease, but the rates of absenteeism, and interpersonal conflicts extratrabalho. Factors related to time and pace of work are very important in determining the suffering psíquico work-related. Long work hours with few breaks for rest and / or meals short, uncomfortable places, night shifts, alternating turns or shifts starting early in the morning, intense rhythms and monotonous; worker's submission to the rhythm of the machines, under which it has no control, pressure from supervisors or bosses for more speed and productivity cause often of anxiety, chronic fatigue and sleep disturbances.

The levels of attention and concentration required to perform the tasks, combined with the level of pressure exerted by the organization of work, can create tension, fatigue and burnout or burn-out g(Translated into Portuguese as burnout syndrome or burnout).

Studies have shown that some heavy metals and solvents may have direct toxic action on the nervous system, causing mental and behavioral changes, manifested by irritability, nervousness, restlessness, impaired memory and cognition, initially not very specific, and Finally, with a chronic course, often disabling and irreversible.

Work accidents may have mental consequences when, for example, affect the central nervous system, as in traumatic brain injury

with concussion and / or injury. The experience of work accidents involving risk to life or limb threatening workers determine sometimes typical psychopathological, psychological syndromes characterized as post-traumatic. Sometimes, there are syndromes related to dysfunction or brain injury, superimposed on psychiatric symptoms, yet blending in to the deterioration of the social network due to changes in the economic landscape of work, exacerbating psychiatric disorders.

Work contexts individuals have been associated with specific psychopathological, which are assigned specific terminology. Seligmann-Silva proposes a characterization for some clinical cases have already been observed. One example is the burn-out, syndrome characterized by emotional exhaustion, depersonalization and self-deprecation. Initially related professions to provide care and assistance to people, especially in economically critical situations and need, the designation has been extended to other professions that involve high emotional investment and personal, in which the work focuses on human problems of high determination and complexities beyond the reach of the worker, such as pain, suffering, injustice, poverty (Seligmann-Silva, 1995).

Another example is the post-traumatic syndromes that refer to experiences of trauma in the workplace in recent times become more frequent, for example, the large number of armed robberies of bank branches with their hostages.

The prevention of mental disorders and behavior related to work based on the procedures for monitoring of health hazards and environments and working conditions. Uses medical knowledge-clinical, epidemiological, occupational hygiene, toxicology, ergonomics, psychology, among other disciplines,

enhances the perception of workers about their work and health and is based on technical standards and regulations involving:

- reconnaissance activities and workplaces where there are chemicals, physical agents, and / or biological risk factors arising from work organization potentially cause disease;
- identifying problems or potential harm to health arising from exposure to identified risk factors;
- identifying and proposing measures to be taken to eliminate or control exposure to risk factors and for protection of workers;
- education and information to workers and employers.

The health surveillance of workers should consider the multiple factors involved in determining the mental and behavioral disorders related to work. In some cases, they are chemical in nature, in others, intrinsically related to the organization and management of work or lack

of work and in many cases result from a synergistic action of these factors.

Upon confirmation of the diagnosis and establish its relationship to work, following the procedures described in Chapter 2, the health services responsible for health care worker should implement the following actions:

- assessing the need for removal (temporary or permanent) worker exposure, sector of employment or work as a whole;

LIST OF MENTAL DISORDERS AND BEHAVIOUR RELATED TO WORK IN ACCORDANCE WITH THE ORDINANCE / MS No. 1.339/1999

- Dementia in other diseases classified elsewhere (F02.8)
- Delirium, not superimposed on dementia, as described (F05.0)
- Mild cognitive impairment (F06.7)
- Organic personality disorder (F07.0)
- Organic mental disorder or symptomatic NS (F09. -)
- Chronic alcoholism (work related) (F10.2)
- Depressive episodes (F32. -)
- State of posttraumatic stress disorder (F43.1)
- Neurasthenia (includes fatigue syndrome) (F48.0)
- Other specified neurotic disorders (including professional neurosis) (F48.8)
- Disorder of sleep-wake cycle due to non-organic factors (F51.2 Nonorganic)
- Feeling of being finished (syndrome burn-out g, the burnout syndrome) (Z73.0)

10.3.1 OTHER DEMENTIA IN SPECIFIC DISEASES CLASSIFIED

OTHER PLACES IN ICD-10 F02.8

1 DEFINITION OF DISEASE - DESCRIPTION

Dementia is conceptualized as a syndrome, usually progressive, chronic disease due to a brain, acquired character, which occur several shortcomings of higher cortical functions, including:

memory, thinking, orientation, comprehension, calculation, learning capacity, language and trial.

Consciousness is not affected and cognitive impairments are accompanied and occasionally preceded by deterioration emotional control, social conduct or motivation (Bertolote, 1997).

May be associated with several diseases that primarily or secondarily affecting the brain, among them, epilepsy, alcoholism, hepatolenticular degeneration, acquired hypothyroidism, lupus erythematosus, trypanosomiasis, poisoning, HIV disease, disease Huntington g's DiseaseParkinson g, the occurrence of multiple infarctions, other ischemic cerebrovascular disease and cerebral contusions repeated, like those suffered by boxers.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Dementia due to drugs and toxins (including dementia due to chronic alcoholism) are 10 to 20% of cases of dementia in general. Head injuries account for 1-5% of cases. No data are available showing the percentages concerning the contribution of work or occupation.

Frames dementia have been found traumatic brain injury (TBI) and the effects of occupational exposure to toxic chemicals following:

- asphyxiating substances: carbon monoxide (CO), hydrogen sulfide (H^2S);
- carbon sulfide;
- heavy metals (Manganese, mercury, lead and arsenic);
- organometallic derivatives (Organotin and tetraethyl lead).

In workers exposed to these neurotoxic chemicals, the diagnosis of dementia

work-related, other causes non-occupational, must be set in Group I of the

Classification Schilling, that work plays the role of necessary cause.

CLINICAL AND DIAGNOSTIC

Diagnostic criteria:

- impairment or disability manifested by cognitive decline (higher cortical), such as learning ability, memory, attention, concentration, language, level of intelligence, problem-solving ability, insight and appropriate social behavior;
- impairment or incapacity for personal activities of daily living.

A decline in cognitive abilities is essential for the diagnosis of dementia. Interference in the performance of social roles within the family, workplace and other spheres of life should not be used as the sole diagnostic criterion or guideline. However, these can serve as indicators

of diagnostic research dementia and, once diagnosed as a useful indicator of severity.

TREATMENT AND OTHER CONDUITS

The approach of dementia patients is characterized by supportive care:

- medical indication for withdrawal of the worker exposure to toxic agent;
- enable patient access to the benefits of the SAT;
- emotional support for patients and their families;
- Symptomatic drug treatment: Benzodiazepines for anxiety and insomnia, antidepressants for depression, antipsychotics for delirium, hallucinations and disruptive behavior;
- management of the work situation: orientation of managers and colleagues about the relationship of mental health problem the patient with the work, seeking collaboration and support for the investigation of other cases in the workplace where the patient / worker comes.

CHAPTER 17 PREVENTION

The prevention of work-related dementia basically consists in monitoring environments, working conditions and the effects or health hazards, as described in the introduction to this chapter.

Requires an integrated, coordinated care across sectors and surveillance, it is desirable that the service is done by a multidisciplinary team, with an interdisciplinary approach, able to deal and support the suffering psíquicogthe worker, the social aspects and intervention in the workplace.

In the presence of a chemical risk factor, the environmental control measures aimed at the elimination or reduction of exposure levels, through:

- entrapment and isolation procedures of work sectors, if possible using hermetically sealed systems;
- hygiene standards and strict security, including exhaust ventilation systems adequate and efficient;
- systematic monitoring of ambient air concentrations;
- adopting forms of work organization that allow reducing the number of exposed workers and exposure time;
- measures of general cleaning of work environments and facilities for personal hygiene, resources for bathing, washing hands, arms, face and exchange of clothing;
- provision by the employer of personal protective equipment appropriate in order to complement measures of collective protection.

The intervention on the conditions of work is based on ergonomic analysis of the actual work or activity, emphasis is, among other things:

- content of tasks, and operational methods of employment;
- pace and intensity of work;
- mechanical factors and physical conditions of employment and production standards;
- shift systems;
- Incentive systems;
- individual psychosocial factors;
- working relationships between colleagues and managers
- and collective protection measures implemented by individual companies;
- the individual and collective strategies adopted by the workers.

Employee participation and managerial levels is essential for the implementation of corrective measures and health promotion involving changes in work organization. Practices that promote health and healthy work environments should include actions for education and prevention of drug abuse, especially alcohol.

The prevention of major accidents with the potential to cause traumatic brain injury should be systematically through management practices and adequate safety measures, with the participation of workers.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate.

DELIRIUM, superimposed on the dementia-NOT AS DESCRIBED ICD-10 F05.0

1 DEFINITION OF DISEASE - DESCRIPTION

Delirium is a syndrome characterized by decreased level of consciousness, with disturbance of orientation (in time and space) and attention (and hipovigilância hipotenacidade) associated with global impairment of cognitive functions. Changes may occur in mood (irritability), perception (illusions and / or visual hallucinations especially) of thought (delusional ideation) and behavior (reactions of fear and agitation). The patient usually presents a characteristic inversion of the sleep-wake schedule with daytime sleepiness and restless sleep. Can be accompanied by neurological symptoms such as tremor, asterixis, nystagmus, incoordination and urinary incontinence. Generally, delirium has a sudden onset (within hours or days), a short course and floating and rapid improvement once the causative factor is identified and corrected. The delirium may occur in the course of a dementia may progress to dementia, to full recovery for morte. Apresenta different levels of severity, from mild to very severe.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Frames delirium have been found between the effects of occupational exposure to the following toxic chemicals or conditions:

- asphyxiating substances: carbon monoxide (CO), hydrogen disulfide (H₂S);

- carbon sulfide;

- heavy metals (manganese, mercury, lead and arsenic);

- organometallic derivatives (Organotin and tetraethyl lead);

- traumatic brain injury (TBI).

Should be ruled out a pre-existing dementia, established or evolving. The delirium may be due to a general medical condition such as that experienced by some patients during periods of hospitalization. 15 to 25% of patients in wards of internal medicine and 30% of patients in surgical wards and intensive care cardiac units have delirium poisoning with substance - and should be checked for specific drug involved or abstinence from substance - or due to multiple etiologies.

In workers exposed to these neurotoxic chemicals, the diagnosis of work-related delirium, excluded other non-occupational causes, can be classified in Group I of the Classification of Schilling in that work plays the role of necessary cause.

3 CLINICAL AND DIAGNOSTIC

With regard to staff related to work, must be observed the following diagnostic criteria:

- decreased level of consciousness - reflected by reduced clarity of awareness about the environment, with reduced capacity to focus, focus, maintain or shift attention. It is the fundamental aspect of the diagnostic criteria for delirium;

- changes in cognition, such as memory deficit, disorientation, language disturbance or disturbance in the development of a perception that is not explained by a preexisting dementia, established or evolving;

- disturbance that develops over a short period of time (hours to days), with a tendency to fluctuation throughout the day;

- there is evidence from history, physical examination or laboratory findings that the disturbance is a direct or indirect, associated with a work situation.

4 TREATMENT AND OTHER CONDUITS

The delirium, as acute or subacute, characterized as a medical emergency, and the first goal of treatment is to control the condition or factor that is causing it. In the case of occupational exposure to toxic agents in the central nervous system, the removal of the patient /

employee desktop is the first action to be taken. Provide physical support (to avoid accidents: patients may be hurt due to disorientation and psychomotor), sensory and environmental control (level of stimulation from the environment: less or more, hold references known to the people calm and patient and family around) .

The symptomatic pharmacological treatment is necessary in cases where there is insomnia, and psychotic symptoms such as hallucinations, delusions and agitation. The drug of choice is haloperidol in the initial dose ranging from 2 to 10 mg (depending on weight, age and physical condition of the patient) intramuscularly, repeated at the end of an hour if the patient remains agitated. The oral dose must be 1, 5 times higher than the parenteral to maintain the same therapeutic effect. The effective daily dose of haloperidol may vary from 5 to 50 mg, according to the severity of delirium. Insomnia can be treated with benzodiazepines of short half-life, such as lorazepam. The use of anticonvulsants is indicated in cases where there is concurrent seizures.

5 PREVENTION

The prevention of delirium, non-overlapping dementia-related work, consists in monitoring environments, working conditions and the effects or health hazards, as described in the introduction to this chapter.

Requires an integrated, coordinated care across sectors and surveillance, it is desirable that the service is done by a multidisciplinary team, with an interdisciplinary approach, able to deal and support the suffering psychiatric worker and the social aspects and intervention in the workplace.

The environmental control measures aimed at eliminating or reducing exposure to chemicals

involved in the genesis of the disease, through:

- entrapment and isolation procedures of work sectors, if possible using systems hermetically sealed;
- hygiene standards and strict security, including exhaust ventilation systems adequate and efficient;
- systematic monitoring of ambient air concentrations;
- adopting forms of work organization that allow reducing the number of exposed workers and exposure time;
- measures of general cleaning of work environments, personal care, resources for bathing, washing hands, arms, face, clothing exchange;
- provision by the employer of personal protective equipment appropriate in order to complement measures of collective protection.

The intervention on the conditions of work is based on ergonomic

analysis of the actual work or activity, emphasis is, among other things:

- content of tasks, and operational methods of employment;
- pace and intensity of work;
- mechanical factors and physical conditions of employment and production standards;
- shift systems;
- Incentive systems;
- psychosocial factors and individual;
- working relationships between colleagues and managers;
- and collective protection measures implemented by individual companies;
- individual and collective strategies adopted by the workers.

Employee participation and managerial levels is essential for the implementation of corrective measures and health promotion involving changes in work organization. Promotion practices

health and healthy work environments should include measures of education and prevention of drug abuse, especially alcohol.

Suspected or confirmed disease compared with the work must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through the instruments themselves, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate

Mild cognitive impairment ICD-10 F06.7

1 DEFINITION OF DISEASE - DESCRIPTION

Mild cognitive impairment characterized by alterations of memory, orientation, learning ability and reduced ability to concentrate on tasks longer periods.

The patient complains of intense feeling of mental fatigue when mental tasks and new learning is perceived subjectively as difficult, although objectively able to do it well. These symptoms may manifest themselves preceding or succeeding frames of various infections (including HIV) or physical disorders, both cerebral and systemic, with no direct evidence of brain impairment.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Frames mild cognitive impairment have been found between the effects of occupational exposure to

following toxic chemicals and physical agents:

- Methyl bromide;
- Lead and its toxic compounds;
- manganese and its toxic compounds;
- mercury and its toxic compounds;
- carbon sulfide;
- toluene aromatic solvents and other neurotoxic;
- Trichloroethylene, tetrachlorethylene, trichloroethane halogenated organic solvents and other neurotoxic;
- other neurotoxic solvents;
- high noise levels.

In workers exposed to these neurotoxic chemicals, among others, the diagnosis of mild cognitive impairment related to work, excluded other non-occupational causes, can be classified in Group I of the Classification of Schilling in that work plays the role of a necessary cause.

CLINICAL AND DIAGNOSTIC

The main symptom is a decline in cognitive performance, including complaints of memory impairment, learning difficulties or concentration. Psychological testing objectives can be useful but should be interpreted with caution, given its specificity and confusion with other causes related to living conditions.

The differential diagnosis with post-encephalitic syndromes and posttraumatic can be done from the etiology and symptoms of lower amplitude, generally lighter and of shorter duration.

TREATMENT AND OTHER CONDUITS

The diagnosis of mild cognitive impairment indicates the existence of injuries to the normal physiology of the cerebral cortex. Whereas the cause of the injury (toxic agent), in the case of mild cognitive impairment related to work, is in the workplace, the main therapeutic measure is the removal of the patient's work.

A thorough evaluation of the stage of cognitive functions should be undertaken by a specialist.

Conductions medical, psychological and social aspects of the case include the pharmacological treatment and symptomatic: Benzodiazepines for insomnia and anxiety, antidepressants for

depression. Psychotic symptoms in cases of mild cognitive impairment are rare. Depending on the degree of dysfunction and / or injury, one can evaluate the usefulness of neuropsychological rehabilitation of patients and rehabilitation professionals.

The control measures and medical surveillance in relation to other workers exposed to toxic agent in the same work environment are critical in preventing further cases.

PREVENTION

The prevention of mild cognitive impairment related to work consists in monitoring environments, working conditions and the effects or health hazards, as described in the introduction to this chapter.

Requires an integrated, coordinated care across sectors and surveillance, it is desirable that the service is done by a multidisciplinary team, with an interdisciplinary approach, able to deal and support the suffering psíquico of the worker and the social aspects and intervention in the workplace.

The environmental control measures aimed at the elimination or reduction of exposure to the chemicals involved in the genesis of the disease, through:

- entrapment and isolation procedures of work sectors, if possible using hermetically sealed systems;
- hygiene standards and strict security, including exhaust ventilation systems adequate and efficient;
- systematic monitoring of ambient air concentrations;
- adopting forms of work organization that allow reducing the number of exposed workers and exposure time;
- measures of general cleaning of work environments, personal care, resources for bathing, washing hands, arms, face and exchange of clothing;
- provision by the employer of personal protective equipment appropriate in order to complement measures of collective protection.

The intervention on the conditions of work is based on ergonomic analysis of the actual work or activity, emphasis is, among other things:

- content of tasks, and operational methods of employment;
- pace and intensity of work;
- mechanical factors and physical conditions of employment and production standards;
- shift systems;
- Incentive systems;

- psychosocial factors and individual;
- working relationships between colleagues and managers;
- and collective protection measures implemented by individual companies;
- the individual and collective strategies adopted by the workers.

Employee participation and managerial levels is essential for the implementation of corrective measures and health promotion involving changes in work organization. Practices that promote health and healthy work environments should include actions for education and prevention of drug abuse, especially alcohol.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

ORGANIC PERSONALITY DISORDER ICD-10 F07.0

1 DEFINITION OF DISEASE - DESCRIPTION

Organic personality disorder is conceptualized as a change in personality and behavior that appears as a residual or concomitant disorder of an illness, injury or brain dysfunction. It is characterized by a significant change in habitual patterns of premorbid behavior, particularly as regards the expression of emotions, needs and impulses. Cognitive function may be compromised in a particular way or even exclusively in the areas of planning and anticipation of the likely social consequences, as in call frontal lobe syndrome, which can occur not only associated with frontal lobe lesions,

but also the lesions of other brain areas circumscribed.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Frames Organic personality disorder have been found between the effects of occupational exposure to toxic chemicals following:

- Methyl bromide;
- Lead or its toxic compounds;

- manganese and its toxic compounds;
- mercury and its toxic compounds;
- carbon sulfide;
- toluene aromatic solvents and other neurotoxic;
- Trichloroethylene, tetrachlorethylene, trichloroethane halogenated organic solvents and other neurotoxic;
- other neurotoxic solvents.

In workers exposed to these neurotoxic chemicals, among others, the diagnosis of organic personality disorder, other causes non-occupational, can be classified in Group I of the Classification of Schilling in that work plays the role of a necessary cause.

3 CLINICAL AND DIAGNOSTIC

In addition to a well-defined history or other evidence of disease or brain dysfunction, a definitive diagnosis requires the presence of two or more of the following:

- consistently reduced ability to persevere in activities with particular purposes, especially those involving longer periods of time and delayed gratification;
- altered emotional behavior, characterized by emotional lability, joy superficial and unmotivated (Euphoria, inappropriate jocularity) and easy change to irritability, rapid bursts of anger and aggression or apathy;
- expression of needs and impulses without considering consequences or social conventions (theft, inappropriate sexual overtures, eating voraciously, or show disregard for personal hygiene);
- cognitive disturbances in the form of suspicion, paranoid ideation and / or preoccupation with a single theme, usually abstract (eg religion, right and wrong);
- remarkable change of speed and flow of language production with aspects such as circumstantiality, prolixity, viscosity and hypergraphia;
- sexual behavior change.

TREATMENT AND OTHER CONDUITS

As the organic personality disorder related to working sequela of brain injury or dysfunction, the treatment aims at the social rehabilitation, or reduce the harm caused by personal behavior and social change.

Pharmacological treatment is symptomatic: Benzodiazepines for insomnia and anxiety, depression and antidepressants to antipsychotics

for disruptive behavior. It can be shown using carbamazepine to control impulsiveness.

Usually there is an indication of disability retirement and rehabilitation measures aimed at the patient's socialization in the family and community.

PREVENTION

The prevention of organic personality disorder related to working consists in monitoring environments, working conditions and the effects or health hazards, as described in the introduction to this chapter.

Requires an integrated, coordinated care across sectors and surveillance, it is desirable that the service is done by a multidisciplinary team, with an interdisciplinary approach, able to deal and support the suffering psíquicogthe worker and the social aspects and intervention in the workplace.

The environmental control measures aimed at the elimination or reduction of exposure to chemicals involved in the genesis of the disease, through:

- entrapment and isolation procedures of work sectors, if possible using hermetically sealed systems;
- hygiene standards and strict security, including exhaust ventilation systems adequate and efficient;
- systematic monitoring of ambient air concentrations;
- adopting forms of work organization that allow reducing the number of workers exposed and the exposure time;
- measures of general cleaning of work environments, personal care, resources for bathing, washing hands, arms, face and exchange of clothing;
- provision by the employer of personal protective equipment appropriate in order to complement measures of collective protection.

The intervention on the conditions of work is based on ergonomic analysis of the actual work or activity in order to know the factors that may contribute to the illness, such as:

- content of tasks, and operational methods of employment;
- pace and intensity of work;
- mechanical factors and physical conditions of employment and production standards;
- shift systems;
- Incentive systems;
- psychosocial factors and individual;

- working relationships between colleagues and managers;
- and collective protection measures implemented by individual companies;
- defense strategies, individual and collective, adopted by the workers.

Employee participation and managerial levels is essential for the implementation of corrective measures and health promotion involving changes in work organization. Practices promotion

health and healthy work environments should include measures of education and prevention of drug abuse, especially alcohol.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union

MENTAL DISORDER OR ORGANIC SYMPTOMATIC NON-SPECIFIED ICD-10 F09 .-

1 DEFINITION OF DISEASE - DESCRIPTION

This term covers a range of mental disorders grouped by having in common a demonstrable etiology in cerebral disease, brain damage or other damage that leads to a dysfunction that may be primary,

as in illness, injury or damage that affects the brain directly and selectively, or secondary, as in systemic diseases in which the brain is one of the multiple organs involved.

This group of the dementia in Alzheimer's disease g,vascular dementia,syndrome

amnésicagorganic (Not induced by alcohol or drugs) and several other organic disorders (hallucinosis, state

catatonic, delusional mood, anxiety), the post-encephalitis syndrome and post-traumatic, including also theorganic psychosis and psychotic symptoms.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Frames organic or symptomatic mental disorder have been found between the effects of exposure

occupational toxic chemicals to the following:

- Methyl bromide;

- Lead and its toxic compounds;
- manganese and its toxic compounds;
- mercury and its toxic compounds;
- carbon sulfide;
- toluene aromatic solvents and other neurotoxic;
- Trichloroethylene, tetrachlorethylene, trichloroethane and other solvents Halogenated organic neurotoxic;
- other neurotoxic solvents.

In workers exposed to these neurotoxic chemicals, among others, the diagnosis of organic mental disorder or symptom, other causes non-occupational, can be classified in Group I of the Classification of Schilling in that work plays the role of a necessary cause.

3 CLINICAL AND DIAGNOSTIC

The clinical picture is characterized by evidence of disease, injury or brain dysfunction or a systemic physical illness, known to be associated with one of syndromes:

- a temporal relationship (weeks or few months) between the development of the underlying disease and the onset of mental syndrome;
- recovery from mental disorders after the removal or improvement of the underlying presumed cause;
- absence of evidence to suggest an alternative cause of mental syndrome, for example, a strong family history or estresse precipitant.

4 TREATMENT AND OTHER CONDUITS

Pharmacological treatment: Benzodiazepines for insomnia and anxiety, depression and antidepressants to antipsychotics for disruptive behavior. It can be shown using carbamazepine to control impulsiveness. Generally, there is an indication of permanent disability, with rehabilitation measures addressing himself more to the patient's socialization in the family and community.

5 PREVENTION

The prevention of organic or symptomatic mental disorder unspecified work-related consists in monitoring environments, working conditions and the effects or health hazards, as described in the introduction to this chapter. Requires an integrated, coordinated care across sectors and surveillance, it is desirable that the service is done by a multidisciplinary team, with an interdisciplinary approach, able to deal and support the suffering psíquico of the worker and the social aspects and intervention in the workplace.

The environmental control measures aimed at the elimination or reduction of exposure to the chemicals involved in the genesis of the disease, through:

- entrapment and isolation procedures of work sectors, if possible using systems

- hermetically sealed;

- hygiene standards and strict security, including exhaust ventilation systems adequate and efficient;

- systematic monitoring of ambient air concentrations;

- adopting forms of work organization that allow reducing the number of workers exposed

- and the exposure time;

- measures of general cleaning of work environments, personal care, resources for bathing, washing hands, arms, face and exchange of clothing;

- provision by the employer of personal protective equipment appropriate in order to complement measures of collective protection.

The intervention on the conditions of work is based on ergonomic analysis of the actual work or activity, emphasis is, among other things:

- content of tasks, and operational methods of employment;

- pace and intensity of work;

- mechanical factors and physical conditions of employment and production standards;

- shift systems;

- Incentive systems;

- psychosocial factors and individual;

- working relationships between colleagues and managers;

- and collective protection measures implemented by individual companies;

- the individual and collective strategies adopted by the workers.

Employee participation and managerial levels is essential for the implementation of corrective measures and health promotion which involve changes in work organization. Practices that promote health and healthy work environments should include actions for education and prevention of drug abuse, especially alcohol.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;

- examine the exposed, to identify other cases;

- notify the case to the systems of health information (Epidemiological, health and / or health worker), through its own

instruments, the DRT / MTE and the labor union

CHRONIC ALCOHOLISM RELATED TO WORK ICD-10 F10.2

1 DEFINITION OF DISEASE - DESCRIPTION

Alcoholism refers to a chronic manner and continued to use alcohol, characterized by lack of regular intake or a pattern of alcohol consumption with frequent episodes of intoxication, and preoccupation with alcohol and their use despite adverse consequences of this behavior for life and health of the user. According to WHO, alcohol dependence syndrome is a work-related problems. The American Society of Addiction in 1990 found the alcoholism as a chronic disease that has its primary development and manifestations influenced by genetic factors, psychosocial and environmental factors, often progressive and fatal. The disruption of control of alcohol be characterized by continuous or periodic and distortions of thought, characteristically denial, that is, the alcoholic drinker tends not to recognize that alcohol abuse causes.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The work is considered one of Psychosocial Risk Factors for chronic alcoholism. The collective consumption of alcoholic beverages associated with employment situations may be due to defensiveness practice as a means to ensure inclusion in the group. It can also be a way to achieve one's own work, due to the pharmacological effects of alcohol own: soothing, euphoric, stimulating, relaxing, sleep inducing, anesthetic and antiseptic. However, these situations are not sufficient to characterize the pathological use of alcohol.

A higher frequency of cases (individual) alcoholism has been observed in certain occupations, especially those that are characterized as being socially discredited and even rejection of some determinants, such as those involving contact with corpses, garbage or waste in general, seizing and killing of dogs, activities in which tension is constant and high, as in dangerous work situations (public transport, banking establishments, construction), high density of mental activity (government offices, banking institutions and commercial), monotonous work, which generates boredom, work on which the person works in isolation from human society (red); work situations that involve prolonged separation from home (Frequent travel, offshore, mining areas).

The relations of chronic alcoholism the work can be classified by ICD-10, using the following codes: "factors influencing health status: (...)

potential health hazards related to socioeconomic and psychosocial circumstances (Z55-Z65 section of CID -10) or the following "additional factors related to the causes of morbidity and mortality classified elsewhere (Y90-Y98 section of ICD-10

The treatment strategies chronic alcoholism include:

PSYCHOTHERAPY: The patient usually has an ambivalent relationship with the therapy sessions and can lose and make a drinking relapse. The therapist must deal with the abuse of alcohol as a psychic defense, be prepared to be tested several times and can not hide behind the lack of motivation when the patient relapses are threatening. Depression, often associated with alcoholism

chronic, can be conducted through the support role of the therapist may be indicated the addition of antidepressant medication. In addition to the experiences of individual psychotherapy, there are experiences of group psychotherapy that can be quite interesting, especially in services

Public health care worker;

PHARMACOLOGICAL TREATMENT: both anti-anxiety drugs such as antidepressants are indicated for the treatment of symptoms of anxiety and depression in patients with disorders related to alcohol abuse;

MUTUAL HELP GROUPS: Alcoholics Anonymous (AA) / Groups for Families of Alcoholics Anonymous (Al-Anon) - the AA groups are voluntary mutual help hundreds of thousands of people with disorders related to alcohol. Founded in the United States (U.S.) in 1935 by two men alcoholics, is widespread throughout the world and exists in Brazil. Although often patients seek to create objections in the AA, when seeking often become enthusiastic participants, which improves the prognosis by increasing adherence to treatment and social support of patients. The service which the employee serves alcohol dependence should deliver this type of referral;

RESOURCE CENTERS OF ATTENTION DAILY after a hospitalization due to alcohol dependence, the return home and community, including work, requires measures of emotional support, guidance and psychosocial rehabilitation phase that may be provided by mental health services, like day care centers.

5 PREVENTION

Prevention actions alcoholism they merely hold courses and lectures for the purpose of attempting to transmit scientific knowledge and advice about the harmful actions of alcohol in the body are often

harmless.

Generally, only positive results reaching programs that identify, and work situations of everyday life, aspects organizationais and environmental risk related to alcohol, trying to implement actions to transform them, for example:

- supervisory practices and leading directly into the worker's dignity and role are considered with special attention in work situations socially discredited;

- provision of adequate facilities, availability of showers and equipment for personal hygiene (including sufficient clothing trade);

- developing strategies to reduce exposure to situations of threats, aggression and anger as popular with the participation of the workers themselves in developing such strategies;

- availability of breaks in pleasant and comfortable, to relieve tension;

- availability of means of communication and interaction with others during the workday in work situations in isolation;

- reduction and control of noise and vibration in the workplace (workers often use alcohol as a hypnotic after working in noisy environments with vibration).

The classic example is that of bus drivers.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;

- examine the exposed, to identify other cases;

- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;

- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;

- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

ICD-10 Depressive Episodes F32 .-

1 DEFINITION OF DISEASE - DESCRIPTION

The depressive episodes characterized by sad mood, loss of interest and pleasure in daily activities, being a common feeling of fatigue increased. The patient may complain of difficulty concentrating, may have low self-esteem and confidence, hopelessness, thoughts of guilt and worthlessness, bleak and pessimistic visions of the future, ideas or suicidal acts. Sleep is often disrupted, usually terminal insomnia. The

patient complains of decreased appetite, weight loss usually sensitive. Symptoms of anxiety are very common. Anxiety tends to be typically more intense in the morning. Changes in psychomotor retardation can range from agitation. There may be a slowness of thought. The depressive episodes should be classified in the mild, moderate, severe without psychotic symptoms, severe with psychotic symptoms.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The ratio of depressive episodes the work can be subtle. The successive disappointments in work situations frustrating losses accumulated over the years of work, the excessive demands for increased performance at work, generated by excessive competition, resulting in constant threat of losing one which the employee is in the hierarchy the company's actual loss, loss of job and dismissal may provide more or less severe depressions or protracted. The unemployment situation has long been associated with development of depressive episodes Several studies in different countries. Some controlled comparative studies have shown a higher prevalence of depression in typists, computer operators, typists,

lawyers, special educators and consultants.

Depressive episodes are also associated with occupational exposure to the following substances

Toxic chemical:

- Methyl bromide;
- Lead and its toxic compounds;
- manganese and its toxic compounds;
- mercury and its toxic compounds;
- carbon sulfide;
- toluene aromatic solvents and other neurotoxic;
- Trichloroethylene, tetrachlorethylene, trichloroethane halogenated organic solvents and other neurotoxic;
- other neurotoxic solvents.

In workers exposed to these neurotoxic chemicals, among others, the diagnosis of

depressive episodes, excluded other non-occupational causes, can be classified in Group I of

Schilling classification, in which work plays the role of necessary cause. Depressive symptoms in these cases usually is not the primary condition and is usually associated with organic mental disorders induced by these substances, such as dementia, delirium, mild cognitive disorder, organic personality disorder, organic mental disorder or symptomatic

unspecified as described in items. So whenever you get a depressive syndrome characterized and there is a history of occupational exposure to toxic substances should be investigated

the coexistence of an organic mental disorder, or indicative of cerebral dysfunction or injury.

3 CLINICAL AND DIAGNOSTIC

The diagnosis of depressive episode requires the presence of at least five of the following symptoms for a period of at least two weeks, and one of the characteristic symptoms is sad mood or diminished interest or pleasure, plus:

- marked loss of interest or pleasure in activities that are normally pleasurable;
- decreased or increased appetite with weight gain or loss (5% or more of body weight in the last month);
- insomnia or hypersomnia;
- psychomotor agitation or retardation;
- fatigue or loss of energy;
- feelings of hopelessness, excessive or inappropriate guilt;
- diminished ability to think and to concentrate or indecisiveness;
- recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan or a suicide attempt or a plan specific suicide.

4 TREATMENT AND OTHER CONDUITS

The limitation of available therapeutic resources depends on the severity and specificity of each case, however, is consensus that treatment of psychiatry depressive episodes involves:

PSYCHOTHERAPY: is indicated even when they are prescribed psychiatric drugs, because the treatment of depressive episode tends to extend for a period of at least six months, in which the patient is frail and in need of emotional support;

PHARMACOLOGICAL TREATMENT: the prescription of antidepressants is indicated depending on the severity of depression. Currently, there are a variety of antidepressant drugs and regimens

possible dose. The prescription should be accompanied by an expert, at least in referral system. Are often indicated Benzodiazepines to control anxiety symptoms

and insomnia early in treatment because the therapeutic effect of

antidepressants begins on average
after two weeks of use;

PSYCHOSOCIAL INTERVENTIONS: one of the central features of depressive episode is discouragement for everyday activities in which the work is included: "a life loses its color and nothing has value."

So sometimes the ability to work is very damaged, preventing the subject of occupational meet their commitments. Often, absences from work are not justified, the first manifestation perceived by relatives or colleagues, bosses or employers. When the depressive episode is related to working conditions this compromise may be earlier and more evident, since the affective factors involved in depression are at work, for example, the loss of a post of leadership or other sudden change in the hierarchy of an organization.

It is very important that the physician or psychiatrist, along with the health team responsible for the patient are able to:

- carefully evaluate the indication of absenteeism through sick leave.

Should involve the patient in that decision, seeking help both away from work if necessary for treatment, and to return to the occupation when recovered;

- justify each of its recommendations to the organization where the patient works, social security and health system, seeking to ensure respect for the clinical situation of the worker;

- help patients cope with the difficulties involved in a process of withdrawal and return to work, for example, the threat of dismissal after the return to work.

Often depressive episodes are moments in the life of the subjects in which major changes take place and where the work is usually involved. This implies often that the subject change positions or jobs or even jobs. Right there, social support is crucial for treatment and to ensure the quality of life. This support is implemented to guarantee the right to

treatment, access to health services, social security and recognition of the suffering. Clinicians and health services can not offer such guarantees, but should trigger these rights and involve organizations and patients;

- guide the patient's relatives, and co-workers, bosses, leaders and managers on how to deal with the situation of the patient's illness, especially as regards the time required for the subject to resume their work capacity. Special attention should be given to the implementation of

decisions, opinions, certificates and issue of CAT, in recognition social (including health insurance and Social Security) from an ailment which, if not physical injury has provable way, is characterized by excessive anxiety compromising the ability to work accurately.

5 PREVENTION

The prevention of depressive episodes related to work basically consists in monitoring environments, working conditions and the effects or health hazards, as described in the introduction to this chapter.

Requires an integrated, coordinated care across sectors and surveillance, it is desirable that the service is done by a multidisciplinary team, with an interdisciplinary approach, able to deal and support the suffering psíquico the worker and the social aspects and intervention in the workplace.

The environmental control measures aimed at eliminating or reducing exposure to chemicals involved in the genesis of the disease, through:

- entrapment and isolation procedures of work sectors, if possible, using hermetically sealed systems;
- hygiene standards and strict security, including exhaust ventilation systems adequate and efficient;
- systematic monitoring of ambient air concentrations;
- adopting forms of work organization that allow reducing the number of exposed workers and exposure time;
- measures of general cleaning of work environments, personal care, resources for bathing, washing hands, arms, face and exchange of clothing;
- provision by the employer of personal protective equipment appropriate in order to complement measures of collective protection.

The intervention on the conditions of work is based on ergonomic analysis of the actual work or activity, emphasis is, among other factors:

- content of tasks, and operational methods of employment;
- pace and intensity of work;
- mechanical factors and physical conditions of employment and production standards;
- shift systems;
- Incentive systems;
- psychosocial factors and individual;
- working relationships between colleagues and managers;
- and collective protection measures implemented by individual companies;

- the individual and collective strategies adopted by the workers.

Sadness and joy are the affection. So, are present in all human relationships, including relationships at work and with work. We feel sad when frustrated in our aspirations, but especially when we lose someone or something that is very dear to us. The sadness he feels at the loss can characterize a state of mourning that is normal or may evolve into a depression (melancholy). The depressive episode related to work characterized by loss of socializing at work: job loss, loss of rank, frustration of expectations related to work and career, noting that the inclusion at work is a fundamental human dimension in our society. The prevention of depression related to work is therefore also ethical. It depends on the economic order and justice in labor relations at both the macro and micro. The classic example of the relationship between depression and economic order is the long-term unemployment.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors;
- accompany the return of the worker to work, whether in the same activity with modifications or restrictions, either to another activity, which is important to ensure that there is progression, recurrence.

STATE OF POST-TRAUMATIC STRESS ICD-10 F43.1

1 DEFINITION OF DISEASE - DESCRIPTION

The state of post-traumatic stress characterized as a late response and / or protracted to a stressful event or situation (short or long term) of exceptionally threatening or catastrophic nature. And, admittedly, would cause extreme distress to any person. Examples: natural disasters or man made, serious accidents, witnessing a violent death or being subjected to torture, rape, terrorism or any other crime. The patient experienced, witnessed or was confronted with an event or events that involved death or threat of death, serious injury or threat of physical integrity to self or others.

Predisposing factors such as personality traits or previous history of neurotic illness may lower the threshold for the development of the

syndrome or aggravate its course, but are neither necessary nor sufficient to explain its occurrence.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The estimated prevalence of disorder, posttraumatic stress in the general population is 1-3%. In risk groups (eg, fighters), prevalence rates vary from 5 to 75%. Not available epidemiological data related to occupations and professions events that pose a risk to sufficiently menacing to trigger the disorder. There are studies restricted to small groups and case reports. The risk of developing disorder, posttraumatic stress related to work seems to be related to hazardous work involving responsibility for human lives, at risk of major accidents, such as work systems in rail, underground and aerial, the work of firefighters, etc.. It is more common in adults and children, but can occur at any age due to the nature of the situations that trigger. The disorder affects more single, divorced, widowed or socially or economically disadvantaged persons.

According to ICD-10, the relationship between disorder, posttraumatic stress Chronic work can be linked to "factors influencing health status: (...) potential health hazards.

socioeconomic and psychosocial circumstances (Z55-Z65 section) or to "additional factors related to the causes of morbidity and mortality classified elsewhere (Y90-Y98 section):

- other physical and mental difficulties related to work: reaction after work accident serious or catastrophic assault on or after work (Z56.6);

- circumstance relating to working conditions (Y96).

In workers who suffered the situations described in the concept of the disease in work circumstances, the diagnosis of disorder, posttraumatic stress, other causes non-occupational, can be classified in Group I of the Classification of Schilling in that work plays the role of a necessary cause.

3 CLINICAL AND DIAGNOSTIC

The typical picture of state of post-traumatic stress includes revivals of repeated episodes of trauma, which bind to a clear knowledge or dreams (nightmares). The patient presents with a persistent feeling of numbness or emotional blunting, decreased involvement or reaction to the world around him, rejecting the activities and situations reminiscent of the episode traumático. Usualmente, there is a state of autonomic arousal with increased hypervigilance, reactions exacerbated incentives and insomnia.

They may also present themselves anxious and depressive symptoms and suicidal ideation. The abuse of alcohol and other drugs can be a complicating factor. May occur and dramatic episodes of acute fear, panic or aggression, triggered by stimuli that evoke a memory and / or sudden revival of trauma or of the original reaction him.

The onset follows the trauma with a latency period that can range from a few weeks

to months (rarely exceeds 6 months). The course is fluctuating but recovery can be expected in most cases. In a small proportion of patients, the chronic condition can progress for many years, becoming a permanent change of personality.

The diagnosis of state of post-traumatic stress can be done in patients with tables

start until six months after an event or period * traumatic stress characterized by:

- stressful event or situation (short or long term) of exceptionally threatening or catastrophic nature, which the patient was exposed in a work situation or work-related;

- recollections or revivals of persistent and recurrent stressor in pictures

thoughts, perceptions or memories vivid and / or nightmares and / or acting or feeling as if the traumatic event were happening again (including the feeling of reliving the experience, illusions, hallucinations and dissociative episodes of flashback, including those that occur on awakening or when intoxicated) and / or distress at exposure to internal or external cues that resemble or symbolize an aspect of the traumatic event and / or physiological reaction or exacerbated the internal evidence

outside that symbolize or resemble an aspect of the traumatic event);

- persistent attitude to avoid circumstances similar or related stressor event (not present before the trauma) as indicated by:

- efforts to avoid thoughts, feelings or conversations associated with trauma;

- efforts to avoid activities, places or people that bring reminders of the trauma;

- inability to recall, either partially or completely, some important aspects of the period of exposure to the stressor;

- significantly diminished interest or participation in significant activities;

- feelings of detachment or estrangement from others;

- emotional distance (eg, inability to have loving feelings);

- sense of future short (for example, no longer expects to have a career, marriage, children, a normal life expectancy); -persistent symptoms of exaggerated alertness;

- difficulty falling asleep or staying asleep;

- irritability or outbursts of anger;

- difficulty concentrating;

- hypervigilance;

- exaggerated response to fear.

* You can make a probable diagnosis if the latency between the event and the onset of symptoms is greater than 6 months. The literature reports that

Latency can be a week or 30 years.

4 TREATMENT AND OTHER CONDUITS

The treatment of disorder, posttraumatic stress involves:

Psychotherapy: individual therapy is always indicated. In the acute phase may follow the model of crisis intervention with support, guidance and development of instruments to deal with the traumatic event. In the chronic phase, psychotherapy aims to let the patient speak freely, letting you recall the traumatic event and, when possible, get to work rebuilding the future. Group psychotherapy may also be effective in preparing the trauma, especially in the chronic phase. Psychotherapy of a family can offer direct support to family members and patient;

PHARMACOLOGICAL TREATMENT: tricyclic antidepressants, especially imipramine and amitriptyline, have a proven action in the treatment of state of post-traumatic stress. The dosage is the same as the treatment of depressive disorders, and the minimum duration of a therapeutic trial should be eight weeks.

Patients who respond well should stay the course of the antidepressant for a minimum period of one year before attempting removal. The use of antipsychotics such as haloperidol, should be avoided except in short periods to control severe aggressiveness or agitation;

PSYCHOSOCIAL INTERVENTIONS: the state of post-traumatic stress related to work usually refers to a traumatic event characterized as a work accident. Psychosocial interventions are the recognition of the relationship of clinical work, the host of the suffering of workers in the workplace and for the guidance and referrals appropriate to the situation. Often, the development of stress disorder, posttraumatic than away from work for treatment, implies a vocational rehabilitation project, since the sequels, especially the persistent phobic pictures, can prevent the return to their previous jobs. The host of desire for change, commitment and efforts

of the healthcare team, to the patient to resume a productive life, are essential for future reconstruction of the affected worker.

5 PREVENTION

The prevention of state of post-traumatic stress related to work involves a complex network of measures for accident prevention, safety and promotion of conditions at work, including work organizational conditions that respect the subjectivity of workers. Requires an integrated, coordinated care across sectors and surveillance, it is desirable that the service is done by a multidisciplinary team, with an interdisciplinary approach, able to deal and support the suffering psíquico the worker and the social aspects and intervention in the workplace.

The intervention on the conditions of work is based on ergonomic analysis of the actual work or activity, emphasis is, among other factors:

- content of tasks, and operational methods of employment;
- pace and intensity of work;
- mechanical factors and physical conditions of employment and production standards;
- shift systems;
- Incentive systems;
- psychosocial factors and individual;
- working relationships between colleagues and managers;
- and collective protection measures implemented by individual companies;
- the individual and collective strategies adopted by the workers.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the decline in health information systems (epidemiological, health and / or health worker), through the instruments themselves, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT for Social Security

•direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

Neurasthenia (Includes Fatigue Syndrome) ICD-10 F48.0 1
DEFINITION OF DISEASE - DESCRIPTION

The most striking feature of fatigue syndrome-related work *is the presence of constant fatigue, accumulated over months or years of work situations where there is no opportunity to obtain necessary and sufficient rest. Fatigue is reported by the patient as being constant, like waking up tired, both physically and mentally, featuring a general fatigue. Other important events include: poor sleep quality, difficulty to deepen sleep, frequent awakenings during the night, especially initial insomnia, difficulty falling asleep or unable to head off ", irritability or lack of patience and discouragement.

Other symptoms that may be part of the syndrome are: headaches, muscle pain (usually in the muscles most used at work), loss of appetite and general malaise. This is generally a chronic condition.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

Complaints of fatigue are extremely common among employed workers. Risk factors of occupational nature that seem to contribute to the emergence of a pathological situation of fatigue are: accelerated pace of work without breaks or pauses without proper conditions for rest and relaxation, long working hours (excessive overtime, commuting from home to work and home from work too long, double shifts to supplement the family income) and working hours in shifts.

Fatigue seems related to the pathological interaction of several of these factors together, over months or years.

These demonstrations have also been associated with occupational exposure to certain chemicals, including:

- Methyl bromide;
- Lead and its toxic compounds;
- manganese and its toxic compounds;
- mercury and its toxic compounds;
- carbon sulfide;
- toluene aromatic solvents and other neurotoxic;
- Trichloroethylene, tetrachlorethylene, trichloroethane halogenated organic solvents and other neurotoxic;
- other neurotoxic solvents.

In workers exposed to work situations described above, the diagnosis of fatigue syndrome

can be included in Group I of the Classification of Schilling, ie, the work acts as a necessary cause.

CLINICAL AND DIAGNOSTIC

The diagnosis of related fatigue syndrome *trabalhogis* made from the occupational history and history of a job well done. One must ask about the duration of working hours, working conditions, rest during the journey, the pace of work, conditions of the labor process (presence of noise and other offenders), work pressure, conditions living and housing, to evaluate the conditions of rest and recreation worker. Do not forget that fatigue syndrome-related work can be found in unemployed workers, it may have been acquired during previous work.

Definitive diagnosis is based on the following criteria:

- persistent and distressing complaints of increased fatigue after mental effort or persistent complaints and overwhelming body of weakness and exhaustion after minimal physical exertion;
- at least two of the following: muscle soreness, dizziness, tension headaches, sleep disturbance, inability to relax, irritability, dyspepsia;
- patient is unable to recover through rest, relaxation or entertainment;
- duration of the disorder at least three months.

The differential diagnosis of fatigue syndrome-related work is based on history and should

be done with:

- fatigue syndrome postviral (G93.9);
- post-encephalitic syndrome (F07.1);
- postconcussion syndrome (F07.2);
- mood disorders (F30 and F39-);
- Panic disorder (F41.0);
- generalized anxiety disorder (F41.1).

4 TREATMENT AND OTHER CONDUITS

It may be advisable to prescribe anxiolytics / hypnotics (benzodiazepines), aiming to control or moderate the most prominent symptoms such as insomnia and irritability, considering always the risk of development of tolerance and dependence of these psychotropic drugs. You should aim to objective changes in working conditions (work organization) and subjective to which a sick worker is subject. The psychotherapy in order to address the subjective changes in relation to work, can be useful.

5 PREVENTION

The prevention of fatigue syndrome-related work consists in

monitoring environments, working conditions and the effects or health hazards, as described in the introduction to this chapter.

Requires an integrated, coordinated care across sectors and surveillance, it is desirable that the service is done by a multidisciplinary team, with an interdisciplinary approach, able to deal and support the suffering psíquicogthe worker and the social aspects and intervention in the workplace.

The environmental control measures aimed at the elimination or reduction of risk factors responsible for the genesis of the disease, present at work, through:

- entrapment and isolation procedures of work sectors, if possible using hermetically sealed systems, where exposure to chemicals and noise, for example;
- hygiene standards and strict security, including exhaust ventilation systems adequate and efficient; mechanization of processes in order to relieve the physical load of work;
- systematic monitoring of concentrations of harmful agents in ambient air;
- adopting forms of work organization to allow diversification of tasks, reducing the isolation of workers and reduce the cognitive demands arising from pressures for productivity, excessive control, among others;
- measures of general cleaning of work environments, comfort and personal hygiene for workers, resources for bathing, washing hands, arms, face and exchange of clothing;
- provision by the employer of personal protective equipment appropriate in order to complement measures of collective protection.

The interventions needed to improve working conditions based on ergonomic analysis of actual work or activity, emphasis is, among other factors:

- content of tasks, and operational methods of employment;
- pace and intensity of work;
- mechanical factors and physical conditions of employment and production standards;
- shift systems;
- Incentive systems;
- psychosocial factors and individual;
- working relationships between colleagues and managers;
- and collective protection measures implemented by individual companies; •individual and collective strategies adopted by the workers.

Employee participation and managerial levels of awareness are essential to the implementation of corrective measures and health promotion involving changes in the organization of trabalho correct the causes of overwork (excessive speed, lack of breaks, etc.). And enable the psychosocial rehabilitation and / or professional employee.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT for Social Security as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

The occurrence of an event of related fatigue syndrome trabalhogshould be considered as a sentinel event, directing the investigation of workplace interventions and psychosocial support the group of workers came from where the stricken;

Other specified neurotic disorders (including Neurosis Pro) CID-10 F48.8

1 DEFINITION OF DISEASE - DESCRIPTION

The group Other specified neurotic disorders includes mixed disorders of conduct, beliefs and emotions who have a close association with a particular culture. According to ICD-10, occupational neurosis, which includes the writer's cramp, * Is included in this group.

The category professional neurosis is defined by Aubert (1993) as "a persistent psychogenic illness, in which symptoms are a symbolic expression of psychic conflict, whose development is tied to a particular situation or professional organization."

The professional neurosis presents three clinical forms:

NEUROSIS CURRENT WORK: traumatic neurosis, related to a current trauma;

Psychoneurosis PROFESSIONAL when a given work situation works as a trigger, reactivating infantile conflicts that remained in the unconscious;

NEUROSIS IN EXCELLENCE: developed from certain

situations that lead to organizational processes of fatigue

(Burn-out *) People who invest heavily in their efforts and ideals given activity.

The category professional neurosis includes work-related psychiatric disorders, in which subjective aspects and personal characteristics, combined with the organizational conditions of work, determine psychological distress *.

Generally, they are paintings of chronic evolution that tend to define as a pattern of behavior.

The organization of work plays a decisive role in the development of these patterns of behavior, to encourage and exploit these personal characteristics.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

According to ICD-10 (Z65 and Z55-section), the relationship of the tables in the group bunched neurosis professional conceptualized as above, with the work can be linked to socioeconomic circumstances and psychosocial

including:

- problems related to employment and unemployment (Z56. -);
- unemployment (Z56.0);
- changing jobs (Z56.1);
- threat of job loss (Z56.2);
- pace of drudgery (Z56.3);
- poor adjustment to work (difficult working conditions) (Z56.5);
- other physical and mental difficulties related to work (Z56.6).

Thus, there is epidemiological evidence of excess prevalence of these disorders in certain occupational groups, their occurrence can be classified as work related disease, the Group II of the Schilling classification, in which the work can be considered as a risk factor in the whole risk factors associated with multifactorial etiology of the disease.

At the current stage of knowledge, this is an epidemiological link, the probabilistic nature, especially when information about working conditions, properly investigated, are consistent with the epidemiological evidence available.

In some cases, the circumstances under which the work is done could cause or contribute to disease recurrence, leading to situate it in Group III of the Classification of Schilling.

3 CLINICAL AND DIAGNOSTIC

The so-called cultural disorders have several features, but two aspects stand out:

- are not easily slotted into the categories of psychiatric classifications established and used internationally;
- were first described in a population or area in particular cultural and subsequently associated with them.

Calls neuroses professionals have been classified in this group of disorders, because there have been no diagnostic criteria established in another classification. The symptoms are nonspecific: fatigue, listlessness, irritability, sleep disturbances (insomnia or excessive sleepiness, etc.).

It is often the inability for work that appears as the first symptom indicating the presence of a professional neurosis: the person who formerly worked well prepared and with dedication can no longer work, feels tired but can not explain the exact reasons. Medical history, including occupational history and physical examination did not reveal somatic determinants for the frame.

The diagnosis is established based on the complaints mentioned in the history of work and analysis of the situation current work. For example, in neurosis of excellence personal values characterized by high standards are crucial to articulate with the organizational culture where excellence is imperative.

4 TREATMENT AND OTHER CONDUITS

When symptoms commit the lives of workers, individual psychotherapy is the best treatment for professional neurosis. It is worth remembering that the indication of psychotherapy involves the subjective implication of who undergoes such treatment. The indication of psychotherapy should be done gently.

5 PREVENTION

The prevention of professional work-related neuroses involves changes in the culture of the organization of work, such as setting restrictions on the operation of individual performance, collective goals that include seeking the welfare of each.

Requires an integrated, coordinated care across sectors and surveillance, it is desirable that the service is done by a multidisciplinary team, with an interdisciplinary approach, able to deal and support the

suffering psíquicogthe worker and the social aspects and intervention in the workplace.

The intervention on the conditions of work is based on ergonomic analysis of the actual work or activity, emphasis is, among other factors:

- content of the tasks, operational methods and work stations;
- pace and intensity of work;
- mechanical factors and physical conditions of employment and production standards;
- shift systems;
- Incentive systems;
- psychosocial factors and individual;
- working relationships between colleagues and managers;
- protection measures implemented by the collective and individual companies;
- the individual and collective strategies adopted by the workers.

Employee participation and managerial levels is essential for the implementation of corrective measures and health promotion which involve changes in work organization.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

The diagnosis of a case professional neurosis should be approached as a sentinel event investigation and indicate the work situation, evaluating the role of work organization in determining the symptomatology. Psychosocial interventions may be indicated to support the group where the affected workers came.

DISORDER OF SLEEP-WAKE CYCLE FACTORS DUE TO NON-ORGANIC ICD-10 F51.2 Nonorganic

1 DEFINITION OF DISEASE - DESCRIPTION

The disorder of sleep-wake cycle due to non-organic factors is defined as a loss of synchrony between the sleep-wake cycle of the

individual sleep-wake cycle and socially established as normal, resulting in insomnia, early interruption of sleep or excessive sleepiness. These disorders can be psychogenic or organic assumed, depending on the relative contribution of psychological, psychosocial or organic.

The disorder of sleep-wake cycle-related work can be included in that category, since, by definition, is given a day's work at night or scheme fixed by the alternation of daytime hours, evening and / or night, under a rotation of shifts.

The shift work is a form of work organization, in which teams of workers take turns to ensure the achievement of the same activity in a project of schedules that differ significantly from normal working hours for the average population. It is considered normal daytime working hours the division of working time in hours between 6 and 18 hours, based on the six-day week and the forty-four hours per week. In shift work, workers perform their activities by modifying their work schedules during the week, month (alternating shifts) or remain on a fixed schedule morning, evening or night. Also considered are the schemes of work shifts and irregular hours of input and output at work every day, week or month.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The ratio of disorder of sleep-wake cycle due to non-organic factors, as conceptualized above, the work can be linked to the "factors influencing health status: (...) potential health hazards related to socioeconomic and psychosocial circumstances (Z55 and Z65-section of ICD-10) or the "additional factors related to the causes of morbidity and mortality elsewhere classified" (Section Y90-Y98 and the ICD-10):

- problems related to employment and unemployment, poor adaptation to the organization of time

- work (shift work or night work) (Z56.6);

- circumstance relating to working conditions (Y96).

Workers who perform their activities in alternating shifts and / or night work, with adjustment difficulties, the diagnosis of disorder of sleep-wake cycle-related work, excluded other non-occupational causes, can be classified in Group I of the Classification of Schilling in that work plays the role of a necessary cause.

In particular cases of workers previously labile or hipersusceptíveis, circumstances such as those described above by ICD-10 could eventually trigger, aggravate or contribute to disease relapse, leading to situate it in Group III of the Classification of Schilling.

3 CLINICAL AND DIAGNOSTIC

Depending on the working hours and night shifts may occur either as an advance late stages of the sleep-wake cycle. These findings may be due to conflicts between the external temporal synchronizers (working hours and other social activities, for example) and internal biological oscillators, the latter responsible for the regulation of biological rhythms. Workers who have one or more of the following complaints:

trouble falling asleep, frequent interruptions in sleep, excessive sleepiness during waking and sleep perception of poor quality should be tested differential diagnosis (PSG) to confirm the absence of sleep disorders not related to work organization.

The code F51.2 Nonorganic is reserved for the disorders of sleep-wake cycle in which psychological factors play the most important role. Cases of suspected organic origin should be classified in G47.2 group, as non-psychogenic disorders of sleep-wake cycle. Thus, the trial if psychological factors are whether or not it is of primary importance to the clinician in each case.

The following clinical features are essential for a definitive diagnosis:

- sleep-wake pattern of the individual out of sync with the desired sleep-wake cycle, which is normal in a given society and private shared by most people in the same environment

cultural;

- as a result of disruption of the sleep-wake cycle, individuals with insomnia during the main period of sleep and excessive sleepiness during waking almost every day for at least a month or recurrently for shorter periods of time;

- quantity, quality and sleep time as unsatisfactory because of marked personal distress or interference with personal functioning in daily life, social or occupational functioning;

- absence of causative organic factor, such as neurological condition or another medical condition, disorder, psychoactive substance use or a drug.

Note: the presence of psychiatric symptoms such as anxiety, depression or hypomania, does not invalidate the diagnosis of a nonorganic disorder of sleep-wake cycle, since this disorder is prevalent in the patient's condition.

4 TREATMENT AND OTHER CONDUITS

Environments are given intra rest days to allow workers to shift the practice of napping during breaks. In severe cases, it is recommended to change working hours.

- It is suggested that the worker to avoid the consumption of substances with caffeine in their composition about 6 hours before the sleep period.

- The practice of regular physical exercise is not exhaustive, should be encouraged. However, these should not be conducted at times near the onset of sleep.

- The intake of foods containing high levels of lipids should be avoided, especially during the night.

- The onset of daytime sleep after night work should be as far as possible, postponed.

- The lower the number of hours between the end of sleep and starting work, the less sleepiness during night work.

- The use of sleep inducing drugs is not recommended.

Shift workers and night workers are subject to greater risk of suffering cardiovascular diseases, gastrointestinal and mental disorders. Therefore, disorders of sleep-wake cycle may be accompanied by other health effects. It is imperative to observe these effects preclude the employee from continuing in their active working life, or the inability to transfer to day work, be entitled to special retirement.

5 PREVENTION

The prevention of disorder of sleep-wake cycle-related work means to organize work so that the shift system is used to a minimum. That is, the economic dimension of shift work should be assessed as having consequences for the health of the worker. The shift system should provide a greater number of hours of rest for workers to recover from fatigue.

On suspicion or diagnosis of a disorder of sleep-wake cycle-related work shall:

- notify the case to the information systems in health, DRT / MTE and the labor union;

- promote medical, psychological and social conditions and changes in working hours on fixed shift;

- promote the company or organization where there is shift work and night, discussions about the organization of work systems in order to implement improvements in shift schedules. Priority should be implemented organizational changes to reduce the number of night shifts and / or the number of days worked irregular hours and the number of

people exposed to conflicts of biological and social synchronizers;

- trigger the surveillance agencies in health: especially SESMT, CIPA, MTE, DRT, unions and state and municipal health programs (outpatient and occupational health).

The prevention of these diseases requires an integrated, coordinated between the health care and surveillance, to ensure that the patient is cared for by a multidisciplinary team, with an interdisciplinary approach that gives an account of both aspects of support to psychological distress the worker and the social aspects and intervention in the workplace.

Employee participation and managerial levels of awareness are essential to the implementation of measures that involve changes in work organization. Practices that promote health and healthy work environments should include education activities and discussions about the organization of work systems in order to implement improvements in shift schedules. Should be applied primarily organizational changes to reduce the number of night shifts and / or the number of days worked irregular hours and the number of people exposed to conflicts of synchronizers biological, and social diagnosis of a case of disorder of sleep-wake cycle-related work should be approached as a sentinel event investigation and indicate the work situation, evaluating the role of work organization in determining the symptomatology. Interventions may be indicated in the shift system, as well as measures of support group where the affected workers came.

SENSE OF BEING FINISHED (SYNDROME BURN-OUT DEPLETION SYNDROME OR PROFESSIONAL) ICD-10 Z73.0

1 DEFINITION OF DISEASE - DESCRIPTION

The feeling of being finished and the burnout syndrome is a kind of prolonged response to chronic emotional and interpersonal stressors at work. Has been described as the result of professional experience in a context of complex social relations, involving the representation that the person has of himself and others. A worker who was once very involved emotionally with their customers, with their patients or the work itself, and wears at a given moment, you give up, loses power or if "Burning" completely. The worker loses the sense of its relationship with work, is not interested, and every effort seemed useless to him.

According to Maslach & Jackson in 1981 and 1986, and Maslach,

in 1993, burnout syndrome comprises three core elements:

- emotional exhaustion (feelings of emotional exhaustion and emotional drain);
 - depersonalization (negative reaction insensitive or excessive deviation of the public who should receive the services or patient care);
 - reduction of personal involvement in work (feeling of reduced competence and success on work).
- Should be a differentiation between burn-out, it would be a response to chronic job stress, other forms of stress response. Syndrome burn-out involves attitudes and negative behaviors with respect to users, customers, organization and work, being a subjective experience that is detrimental to practical and emotional for the worker and the organization. The traditional picture of stress does not involve such attitudes and conduct, is an exhaustion that interferes with the personal lives of individuals, but not so direct in its relationship with work.

May be associated with an increased susceptibility to physical illness, alcohol or other drugs (to obtain relief) and for suicide.

EPIDEMIOLOGY 2 - RISK FACTORS OF OCCUPATIONAL NATURE KNOWN

The syndrome affects mainly professionals or carers services, when in contact directly with users, employees of education, health, police, social workers, prison officers, teachers, and others.

Lately, have been reported increases in prevalence of burnout syndrome workers from work environments that undergo organizational changes, such as job layoffs reduced work week, no replacement of substitutes, and downsizing (downsizing) call in restructuring.

The risk of burnout syndrome is greater for those living the threat of mandatory changes in working hours and significant decline in economic status. All factors of social and economic insecurity increases the risk (incidence) of burnout in all age groups.

In general, factors related to work are more strongly related to the work itself than with the factors biographical or personal. The most important predisposing factors are: role conflict, loss of control or autonomy and lack of social support.

The ratio of burn-out syndrome for burnout with the work, according to ICD-10, may be linked to the "factors influencing health status: (...) potential health hazards related to socioeconomic and psychosocial circumstances (Z55-Z65 Section of the ICD-10):

- pace of drudgery (Z56.3);

- other physical and mental difficulties related to work (Z56.6).

If there is epidemiological evidence of the incidence of the syndrome in certain occupational groups, their occurrence can be classified as work related disease, the Group II of the Classification of Schilling.

The work can be considered a risk factor in the set of risk factors associated with etiology multicausal desta disease. This is an epidemiological link, the probabilistic nature, especially when information about working conditions, properly investigated, are consistent with the epidemiological evidence available.

3 CLINICAL AND DIAGNOSTIC

The clinical picture can be identified:

- history of high subjective involvement with work, employment, profession or undertaking given, it often gains the character of the mission;
- feelings of wear emotional emptiness and affective (emotional exhaustion);
- complaints of negative reaction insensitive or excessive deviation of the public who should receive the services or patient care (depersonalization);
- complaining of feeling of reduced competence and success at work.

Generally, these are associated with nonspecific symptoms such as insomnia, fatigue, irritability, sadness, indifference, apathy, anxiety, tremors and restlessness, characterizing depressive syndrome and / or anxious. The diagnosis of these syndromes associated with the completion of the above criteria leads to a diagnosis of burnout syndrome.

4 TREATMENT AND OTHER CONDUITS

The treatment of burnout syndrome involves psychotherapy, pharmacological and psychosocial interventions. However, the intensity of the prescription of each of the therapeutic resources depends on the severity and specificity of each case.

PSYCHOTHERAPY: Psychotherapy is indicated even when they are prescribed psychiatric drugs, because the burnout syndrome refers to a process of disinvestment in the emotional work that had been the object of all or much of that investment. The patient therefore needs time and space to rethink (and resign ourselves)

their integration at work and in life. The patient is frail and in need of emotional support;

PHARMACOLOGICAL TREATMENT: the prescription of antidepressants or anxiolytics is indicated according to the presence and severity of depressive and anxiety symptoms. Currently there are a variety of antidepressant drugs and regimens available. The prescription should be accompanied by an expert, at least in referral system. Often, they are indicated Benzodiazepines to control symptoms of anxiety and insomnia, early treatment, because the therapeutic effect of antidepressants begins, on average, after two weeks of use;

PSYCHOSOCIAL INTERVENTIONS: one of the central features of burnout syndrome is the removal of the affective work, compromising the performance, and often their own ability to work. The physician should carefully evaluate the indication of absenteeism through sick leave. The physician should involve the patient in that decision, trying to help you both away from work if necessary for treatment, and to return to work when recovered.

In addition, the physician and other members of the healthcare team should be able to justify each of its recommendations to the organization where the patient works, social security and health system, seeking to ensure respect for the clinical situation of the worker. The doctor should be able to cope with the difficulties involved in a process of withdrawal and return to work, for example, the threat of dismissal after the return to work.

Often, the burnout syndrome featuring a moment of life of the subject in great changes that take place position, the post

Working in the hierarchy or even employment. Sometimes burnout syndrome a sequel is found in a patient unemployed. These situations require social support, crucial to ensuring the quality of life achieved in

securing the right to treatment, access to health services, social security and recognition of the suffering. The health team should be able to counsel patients and their families about these rights and guide family members, coworkers, bosses, bosses and managers to deal with the situation of the patient's illness until he resume his work capacity *. Special attention should be given to the implementation of decisions, opinions, certificates and issue of CAT, in recognition social (including health insurance and / or Social Security) from an ailment that, even though they showed apparent injury, compromises the ability working.

5 PREVENTION

The prevention of burnout syndrome involves changes in the culture of the organization of work, establishing restrictions on the operation of individual performance, decreased labor intensity, reduced competitiveness, seeking collective goals that include the welfare of each. The prevention of these diseases requires an integrated, coordinated between the health care and surveillance. It is important that the patient is cared for by a multidisciplinary team, with interdisciplinary approach that addresses both aspects of support for psychological distress the worker and the social aspects and intervention in the workplace.

Suspected or confirmed disease compared with the work, you must:

- inform the employee;
- examine the exposed, to identify other cases;
- notify the case to the systems of health information (epidemiological, health and / or health worker), through its own instruments, the DRT / MTE and the labor union;
- provide for the issuance of CAT, where the worker is insured by the SAT of Social Security, as described in Chapter 5;
- direct the employer to adopt the technical and managerial procedures to eliminate or control risk factors.

* Among the health problems, mental disorders are responsible for as many days off work, or That is, health problems that are

workers away from work for long periods. Moreover, as this is not a visible injury or a measurable physical process, often patients do not have the legitimately recognized their suffering.

The diagnosis of a case burnout syndrome should be approached as a sentinel event investigation and indicate the work situation, evaluating the role of work organization in determining the symptomatology. Interventions may be indicated in the work

organization, as well as measures of support group where the affected workers came.

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TABLES

Diagnosis and Management of Work Related Diseases: Manual of Procedures for the Health Services Ministry of Health - PAHO

ANNEX

LIST OF DISEASES RELATED TO THE WORK OF THE MINISTRY OF HEALTH - (Prepared in accordance with Law 8080/90 - section VII, paragraph 3 of Article 6 - prepared according to the taxonomy, nomenclature and coding of ICD-10)

Ordinance No. 1339/GM on November 18, 1999.

The Minister of State for Health, in exercise of its functions, and considering Article 6, paragraph 3 of section VII of Law No. 8.080/90, which delegates to the National Health System - SUS periodic review of the official list of conditions originating in the process work, the resolution of the National Health Council, No. 220, dated May 5, 1997, recommending that the Ministry of Health to publish the list of diseases related to work, the importance of defining the disease profile of the working population to establish public policies in health worker decides:

Article 1 Establishing the List of Diseases Related to Work, to be used as a reference of the diseases that originate in the work process in the Health System for clinical use and

Epidemiological, contained in Annex I to this Ordinance.

Article 2 This list may be revised annually.

3 This Ordinance shall enter into force upon its publication.

Minister Jose Serra

Decree No. 3048 of May 6, 1999

REGULATION OF SOCIAL SECURITY

ANNEX IV

CLASSIFICATION OF HARMFUL AGENTS

REGULATION OF SOCIAL SECURITY

N E X O II

PATHOGENS CAUSING DISEASES OR PROFESSIONAL WORK, AS PROVIDED IN ART. 20 THE LAW In 8213, 1991 (Writing by Decree No. 6957 of September 9, 2009)

CLASSIFICATION OF HARMFUL AGENTS

CODE	NOXIOUS AGENT	TIME EXPOSURE
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1.0.0	<p>CHEMICAL AGENTS</p> <p>What determines the benefit is the presence of the agent in the production process and its realization in the workplace, in condition (concentration) can cause damage to health or integrity física. As listed activities are exemplified in which there may be exposure.</p> <p>What determines the entitlement to benefit is the worker's exposure to harmful agent present in the work environment and production process at the level of concentration exceeding the tolerance limits set. (Writing by Decree, No. 3265, 1999)</p> <p>The list of harmful agents is comprehensive, while the listed activities in which there may be exposure, is illustrative. (Writing by Decree, No. 3265, 1999)</p>	
1.0.1	<p>ARSENIC AND ITS COMPOUNDS</p> <p>a) extraction of arsenic and its toxic compounds; b) arsenic metal ores; c) use of hydrogen arsenic (arsine) in organic synthesis and processing of electronic components; d) preparation and manufacture of paints and lacquers; e) manufacture, preparation and application of insecticides, herbicides, rodenticides and fungicidal with the use of arsenic compounds; f) production of glass, lead alloys and medicines with the use of arsenic compounds; g) conservation and fur tanning, wood preservation and treatment with the use of arsenic compounds.</p>	25 YEARS
1.0.2	<p>ASBESTOS</p> <p>a) extraction, processing and handling amiantíferas rocks; b) manufacture of linings for brakes, clutches and insulating materials containing asbestos; c) manufacture of cement; d) blending, carding, spinning and weaving of asbestos fibers.</p>	20 YEARS
1.0.3	<p>BENZENE AND ITS TOXIC COMPOUNDS</p> <p>a) production and processing of benzene; b) use of benzene as a raw material in organic synthesis and production of derivatives; c) use of benzene as a feedstock in the extraction of</p>	25 YEARS

	vegetable oils and alcohols; d) use of products containing benzene, such as glues, paints, varnishes, solvents and graphics; e) production and use of chlorobenzenes and derivatives; f) manufacture and vulcanization of rubber; g) manufacturing and retreading of tires.	
1.0.4	TOXIC BERYLLIUM AND ITS COMPOUNDS a) mining, milling and processing of beryllium; b) manufacture of chemical compounds and alloys of beryllium; c) manufacture of fluorescent tubes and ampoules of X-ray; d) production of burnt f) use of beryllium in the aerospace industry.	25 YEARS
1.0.5	BROMINE AND ITS TOXIC COMPOUNDS a) manufacture and use of bromine and acid bromico.	25 YEARS
1.0.6	CADMIUM AND ITS TOXIC COMPOUNDS a) extraction, processing and preparation of cadmium alloys; b) manufacturing cadmium compounds; c) use of cadmium in welding electrodes; d) use of cadmium in the electrolytic coating of metals; e) use of cadmium as a pigment and stabilizer in the plastic industry; f) fabricating electrodes of alkaline batteries with nickel-cadmium.	25 YEARS
1.0.7	COAL AND THEIR DERIVATIVES a) extraction, manufacturing, processing and utilization of coal, tar, tar, bitumen and pitch; b) extraction, production and use of mineral oils and paraffins; c) extraction and utilization of anthracene and carbon black; d) production of coke.	25 YEARS
1.0.8	LEAD AND ITS TOXIC COMPOUNDS a) extraction and processing of lead ore; b) metallurgy and manufacture of alloys and lead compounds;	25 YEARS

	<p>c) manufacturing and reform of electric accumulators; d) manufacture and use of lead-tetraethyl lead and tetramethyl; e) the manufacture of paints, enamels and varnishes based on lead compounds; f) Spray painting using paint with lead pigments; g) manufacture of objects and artifacts of lead and its alloys; h) litharge by vulcanization of rubber or other lead compounds; i) use of lead in soldering processes; j) the manufacture of glass, crystal and vitreous enamel; l) the manufacture of artificial pearls; m) manufacture and use of lead-based additives for the plastics industry.</p>	
1.0.9	<p>CHLORINE AND ITS TOXIC COMPOUNDS a) manufacture and use of organochlorine pesticides; b) manufacture and use of chloroethylamines (nitrogen mustard); c) manufacture and handling of polychlorinated biphenyls (PCBs); d) manufacture and use of vinyl chloride monomer as in the manufacture of polyvinyl chloride (PVC) and other resins and as an intermediate in chemical production or as an organic solvent; e) production of chloroprene; f) manufacture and use of chloroform (trichloromethane) and carbon tetrachloride.</p>	25 YEARS
1.0.10	<p>CHROME AND ITS TOXIC COMPOUNDS a) manufacturing, industrial employment, manipulation of chromium, chromic acid, chromate and dichromate; b) manufacturing of iron-chromium alloys; c) electrolytic coating of metal surfaces and polished chrome; d) paint spraying using chrome pigment inks; e) welding of stainless steel.</p>	25 YEARS
1.0.11	<p>Carbon Disulfide a) manufacture and use of carbon disulfide; b) manufacture of artificial silk and viscose (rayon); c) manufacture and use of solvents, pesticides and</p>	25 YEARS

	herbicides containing carbon disulfide; d) the manufacture of varnishes, resins, salts of ammonia, carbon tetrachloride, optical glass and textiles with the use of carbon disulfide.	
1.0.12	PHOSPHORUS AND ITS TOXIC COMPOUNDS a) extraction and preparation of phosphorous and its compounds; b) manufacturing and application of phosphorus and organophosphorus products (organic synthesis, fertilizer and pesticides); c) manufacture of munitions and explosive weapons.	25 YEARS
1.0.13	IODINE a) manufacturing and industrial employment of iodine.	25 YEARS
1.0.14	MANGANESE AND ITS COMPOUNDS a) extraction and processing of manganese ores; b) production of manganese alloys and compounds; c) manufacture of dry batteries and accumulators; d) preparation of potassium permanganate and dyes; e) the manufacture of specialty glass and ceramics; f) use of electrodes containing manganese; g) manufacture of paints and fertilizers.	25 YEARS
1.0.15	MERCURY AND ITS COMPOUNDS a) mining and mercury use and manufacture of its compounds; b) manufacture of fuses with fulminate of mercury; c) manufacture of paints with pigment containing mercury; d) the manufacture and maintenance of gauges and laboratory; e) manufacture of light bulbs, vacuum tubes and ampoules of X-ray; f) manufacturing of switches, batteries and rectifiers current; g) used as catalyst and electrolysis; h) gilding, silvering, tinning tanning and mirrors and metals; i) raising and tanning leather and wood preservation; j) recovering the mercury;	25 YEARS

Tratado de Toxicologia Ocupacional

	l) the amalgamation of zinc. m) heat treatment of metal amalgams; n) manufacture and application of fungicides.	
1.0.16	NICKEL AND ITS TOXIC COMPOUNDS a) extraction and processing of nickel; b) nickel metal; c) manufacture of nickel-cadmium.	25 YEARS
1.0.17	Oil, oil shale, NATURAL GAS AND ITS DERIVATIVES a) extraction, processing, manufacturing and maintenance activities carried out in units of extraction, oil and petrochemical plants; b) processing and application of asphalt mixtures containing polycyclic.	25 YEARS
1.0.18	FREE SILICA a) extraction of minerals in the open; b) processing and processing of mineral products that generate dust containing free silica crystallized; c) treating, etching and cleaning of metals and glass matting with sandblasting; d) manufacturing, processing, retrieval and application of refractory materials; e) manufacture of grindstones, grinding wheels and polishing pastes and powders; f) making glass and ceramics; g) construction of tunnels; h) dry cutting and grinding materials containing silica.	25 YEARS
1.0.19	OTHER CHEMICALS GROUP I - STYRENE; butadiene-styrene, Acrylonitrile, 3.1 Butadiene, Chloroprene, mercaptans, n-hexane, toluene diisocyanate (TDI) AROMATIC AMINES a) manufacture and vulcanization of rubber; b) manufacturing and retreading tires. GROUP II - AROMATIC AMINES, aminobiphenyl, auramin, AZATHIOPRINE, BIS (CHLORINE METHYL) ETHER, 1-4 butanediol, DIMETANOSULFONATO (MILERAN), CYCLOPHOSPHAMIDE, CLOROAMBUCIL, DIETILESTIL-BESTROL, ACRONITRILA,	25 YEARS

	<p>NITRONAFTILAMINA 4-DIMETHYL-aminoazobenzene, benzopyrene, BETA- propiolactone, BISCLOROETILETER, BISCLOROMETIL, CLOROMETILETER, DIANIZIDINA, dichlorobenzidine, DIETILSULFATO, dimethyl sulfate, ethylene amines, ethylenethiourea, phenacetin, methyl iodide ETILNITROSURÉIAS, METHYLENE-ORTOCOLOROANILINA (MOCA), nitrosamines, orthotoluidine, Oxime-Talon, procabazine, PROPANOSULTONA, 1-3 -butadiene, ethylene oxide, ESTILBENZENO, toluene diisocyanate (TDI), creosote, 4-AMINODIFENIL, benzidine, BETANAFTILAMINA, STYRENE, 1-chloro-2, 4 - nitrodiphenyl, 3-POXIPRO-CLOTH</p> <p>a) Manufacture of magenta (aniline and orthotoluidine); b) manufacture of synthetic fibers; c) chemical synthesis; d) manufacture of rubber and foam; e) the manufacture of plastics; f) production of medicines; g) operations to preserve the wood with creosote; h) sterilization of surgical materials.</p>	
2.0.0	<p>PHYSICAL AGENTS</p> <p>Exposure above the limits of tolerance specified or the activities described.</p>	
2.0.1	<p>NOISE</p> <p>a) continuous exposure to noise above 90 decibels. a) exposure to Normalized Exposure Levels (NEN) up to 85 dB (A). (Writing by Decree No. 4882 of 2003)</p>	25 YEARS
2.0.2	<p>VIBRATIONS</p> <p>a) work with jackhammers and pneumatic hammers.</p>	25 YEARS
2.0.3	<p>IONIZING RADIATION</p> <p>a) extraction and processing of radioactive minerals; b) activities in mining with exposure to radon; c) execution of maintenance and supervision in units of extraction, treatment and processing of radioactive minerals with exposure to ionizing radiation; d) operations with nuclear reactors or radioactive</p>	25 YEARS

Tratado de Toxicologia Ocupacional

	sources; e) work done on exposure to ultraviolet Alpha, Beta, Gamma and X, the neutrons and radioactive substances for industrial, therapeutic and diagnostics; f) the manufacture and manipulation of radioactive products; g) research and studies with ionizing radiation in laboratories.	
2.0.4	ABNORMAL TEMPERATURES a) work with exposure to heat above the tolerance limits established in NR-15, Ordinance No. 3.214/78.	25 YEARS
2.0.5	ABNORMAL ATMOSPHERIC a) work in coffins or hyperbaric chambers; b) work in caissons or tunnel under compressed air; c) diving operations with the use of riot gear or other equipment.	25 YEARS
3.0.0	BIOLOGICAL Exposure to these agents only in related activities.	
3.0.1	MICROORGANISMS AND PARASITES INFECTIOUS AND LIVING TOXINS PARASITES AND INFECTIOUS MICROORGANISMS-CONTAGIOUS LIVING AND TOXINS (Writing by Decree No. 4882 of 2003) a) work in health facilities in contact with patients with infectious diseases or handling of contaminated materials; b) work with infected animals for treatment or for the preparation of serum, vaccines and other products; c) work in laboratories autopsy, anatomic anatomy and histology; d) work of exhuming bodies and animal waste handling deteriorated; e) work in galleries, pits and tanks, sewage; f) emptying the digesters; g) garbage collection and industrialization.	25 YEARS
4.0.0	ASSOCIATION OF AGENTESExposição agents matched exclusively in the activities specified. ASSOCIATION OF STAFF (Writing by Decree No. 4882 of 2003)	

	In the associations of agents that are above the tolerance level will be considered on the framework that require less exposure time. (Writing by Decree No. 4882 of 2003)	
4.0.1	PHYSICAL, CHEMICAL AND BIOLOGICAL a) underground mining activities which are carried away from the fronts of production.	20 YEARS
4.0.2	PHYSICAL, CHEMICAL AND BIOLOGICAL a) work on ongoing activities in the underground of underground mines in front of production.	15 YEARS

REGULATION OF SOCIAL SECURITY

N E X O II

PATHOGENS CAUSING DISEASES OR PROFESSIONAL WORK, AS PROVIDED IN ART. 20 OF LAW NO 8213 OF 1991

(Writing by Decree No. 6957 of September 9, 2009)

PATHOGENS	WORKS CONTAINING THE RISK
CHEMICALS	
I - Arsenic and arsenic compounds	arsenical ore metallurgy and electronics industry; Extraction and preparation of arsenic compounds; manufacture, formulation and use of paints, lacquers (arsine gas), insecticidal, fungicidal and rodenticides; industrial processes where there is hydrogen evolution arsenic; preparation and storage of fur and feathers (stuffing animals) and wood preservation; agents in the production of glass, lead alloys, pharmaceuticals and semiconductors.
II - ASBESTOS OR ASBESTOS	amiantíferas extraction of rocks, drilling, cutting, dismantling, crushing, screening and handling; dumps the material from the extraction, crushing; blending, carding, spinning and weaving of asbestos; manufacture of brake linings, insulation materials and asbestos cement products; any setting or demolition of asbestos products that produce airborne particles of asbestos.

III - BENZENE OR ITS TOXIC COUNTERPARTS	<p>Manufacture and use of benzene, its homologues or their derivatives and amino nitrous: petrochemical plants which produce benzene; chemical industry or in laboratories; production of synthetic glue; users of synthetic glue for the manufacture of footwear, leather or rubber and furniture; production of paints; printers (especially in photogravure); paint gun; welding.</p>
IV - BERYLLIUM AND ITS TOXIC COMPOUNDS	<p>extraction, crushing and processing of beryllium; manufacturing and foundry alloys and compounds;</p> <p>) use in aerospace and manufacturing of precision instruments and computers, cutting tools non-sparking for the oil industry;</p> <p>) manufacture of fluorescent tubes, ampoules of X-rays, vacuum electrodes, cathodes burners and moderators in nuclear reactors; manufacture of crucibles, special glass and porcelain insulators.</p>
V - BROMINE	<p>Manufacture and use of bromine and acid brômico.</p>
VI - CADMIUM OR THEIR COMPOUNDS	<p>extraction, processing, preparation and casting of alloys;</p> <p>manufacturing cadmium compounds for welding; welding;</p> <p>use in metal coating (galvanizing), as pigments and stabilizers in plastics, in nickel-cadmium and silver soldering.</p>
VII - THE TUNGSTEN SINTERED metal carbides	<p>Production of sintered carbides (mixing, spraying, patterned, heating furnace, fit, precision spraying), the manufacture of tools and components for machines and sharpening tools. Workers in the immediate vicinity and within the same workshop.</p>
VIII - LEAD OR ITS TOXIC COMPOUNDS	<p>mineral mining, metallurgy and refining of lead; manufacture of batteries and accumulators (plates);</p>

	<p>manufacture and use of lead-tetraethyl lead and tetramethyl;</p> <p>manufacture and application of paints, enamels and varnishes based on lead compounds;</p> <p>casting and rolling of lead, brass, etc.;</p> <p>manufacture or handling of lead compounds and alloys;</p> <p>manufacture of objects and artifacts of lead, including ammunition;</p> <p>vulcanization of rubber by litharge or other lead compounds;</p> <p>welding;</p> <p>printing industry;</p> <p>glass manufacturing, glass and vitreous enamel;</p> <p>scrap, scrap metal;</p> <p>manufacture of artificial pearls;</p> <p>pottery;</p> <p>manufacture of matches.</p>
IX - CHLORINE	Manufacture and use of chlorine and hydrochloric acid.
X - CHROME OR ITS TOXIC COMPOUNDS	<p>manufacture of chromic acid, chromate and dichromate and chromium alloys;</p> <p>electrolytic metal plating (electroplating);</p> <p>tanning and other work with the leather;</p> <p>paint gun with pigments chromium compounds, polishing furniture;</p> <p>handling of chromic acid, chromate and dichromate;</p> <p>welding of stainless steel;</p> <p>manufacture of cement and construction work;</p> <p>printing and photographic technique.</p>
XI - FLUORIDE OR ITS TOXIC COMPOUNDS	<p>manufacture and use of fluoride and hydrofluoric acid;</p> <p>Steel (as fluxes);</p> <p>manufacturing, tiles, ceramic, cement, glass, enamel, glass fiber, phosphate fertilizers;</p> <p>gasoline production (such as alkylating catalyst);</p> <p>electric welding;</p> <p>electroplating;</p>

	heating surfaces; system of rocket fuel.
XII - MATCH OR ITS TOXIC COMPOUNDS	Extraction and preparation of phosphorous and its compounds; manufacturing and application of phosphorus and organophosphorus products (organic synthesis, fertilizer, pesticides); manufacture of incendiary projectiles, explosives and asphyxiating phosphor-based white; manufacture of bronze alloys; sprayers, agricultural workers and responsible for the storage, transport and distribution of organophosphate pesticides.
XIII - AROMATIC OR ALIPHATIC HYDROCARBONS (Their halogenated toxic)	
- Methyl chloride	Chemical synthesis (methylation), soda, special agent for extraction.
- Methylene chloride	Solvent (oils, greases, waxes, cellulose acetate), degreaser, paint remover.
- Chloroform	Solvent (lakes), the extraction agent.
- Carbon Tetrachloride	Chemical synthesis, fire extinguishers.
- Ethyl Chloride	Chemical synthesis, local anesthetic (cooling).
1.1 - Dichloroethane	Chemical synthesis, solvent (resins, rubber, asphalt, paints), degreaser.
1.1.1 - Trichloroethane	Degreasing agent for metal cleaning and dry cleaning.
1.1.2 - Trichloroethane	Solvent.
1.2 - dibromoethane	Solvent.
	Degreasing agent, dry cleaning and extraction, chemical synthesis.
	Degreasing agent, dry cleaning and extraction, chemical synthesis.
	Intermediate in the manufacture of polyvinyl chloride.
	Insecticide spraying (crops), chemical synthesis.
	Chemical synthesis, a special agent for extraction.
	Insecticide spraying (soils), fire extinguisher,

	solvent (celluloid, grease, oil, waxes). Chemical synthesis, solvent. Chemical synthesis, solvent.
XIV - IODINE	Manufacture and use of iodine.
XV - MANGANESE AND ITS COMPOUNDS TOXIC	extraction, processing and grinding of pyrolusite (manganese dioxide); production of manganese alloys and compounds; Steel; manufacture of dry batteries and accumulators; preparation of potassium permanganate and the manufacture of dyes; manufacture of specialty glass and ceramics; welding with electrodes containing manganese; manufacture of paints and fertilizers; 9. leather tanning.
XVI - MERCURY AND ITS TOXIC COMPOUNDS	mineral extraction and manufacture of mercury and its compounds; manufacture of fuses with fulminate of mercury; manufacture of paints; manufacture of welding; manufacturing apparatus: manometers, thermometers, switches, lamps, vacuum tubes, X-ray bulbs, rectifiers; amalgamation to manufacture zinc electrodes, batteries; tin and gilt mirrors; stuffing animals with mercury salts; recovery of mercury by distillation of industrial waste; heat treatment of amalgam of gold and silver recovery of these metals; secretagem hair, horsehair and feathers, and raising the base of mercury compounds; fungicide seed treatment and glows in the protection of plants and wood.
XVII - SUBSTANCES Asphyxiating 1. Carbon monoxide	Production and distribution of gas obtained from solid fuels (coal gasification), motor mechanics, especially gasoline-powered, semi-enclosed in enclosures, arc welding and acetylene, boilers, chemical industry, metallurgy, foundry,

Tratado de Toxicologia Ocupacional

	underground mining, use of explosives; fire control, traffic control, construction of tunnels; breweries.
2. Hydrogen cyanide or its toxic derivatives	Insecticide spraying operations, synthesis of organic chemicals; eletrogalvanoplastia; extracting gold and silver, production of steel and plastic (especially acrylonitrile-styrene); steel (coke ovens).
3. Hydrogen sulfide (hydrogen sulphide)	Stations, wastewater treatment, mining, metallurgy; work in silos, sugar beet processing, tanneries and slaughterhouses, production of viscose and cellophane, chemical industry (production of sulfuric acid, barium salts), tunneling, drilling oil and gas wells; carbonization of coal at low temperature; lithography and photoengraving.
XVIII - FREE SILICA (Silicon oxide - Si O ₂)	<p>extraction of minerals (work underground and open pit);</p> <p>stripping, metal cleaning, foscamento glass with sandblasting, and other activities in which sand is used as an abrasive;</p> <p>) manufacture of refractory material for furnaces, fireplaces and hearths, waste recovery;</p> <p>) manufacture of millstones, grindstones, scouring powders, powder or paste for polishing metal;</p> <p>grinding and handling of silica in the glass industry and porcelain;</p> <p>) quarrying;</p> <p>i) work in tunnel construction;</p> <p>ii) thinning and polishing of stones.</p>
XIX - SULFIDE OR CARBON Carbon Disulfide	<p>manufacture of carbon sulfide;</p> <p>industry viscose rayon (artificial silk);</p> <p>manufacture and use of solvents, insecticides, fungicidal and herbicides;</p> <p>manufacture of varnishes, resins, salts of ammonia, carbon tetrachloride, textiles, electronic vacuum tubes, grease;</p> <p>dry cleaning, electroplating, fumigation of grains;</p> <p>processing oil, sulfur, bromine, wax, grease and</p>

	iodine.
XX - tar, BITUMEN, COAL MINING, AND PRODUCTS OR WAX WASTE OF THESE SUBSTANCES, epitheliomas CAUSING EARLY SKIN	Processes and industrial operations or not, they are used tar, pitch, bitumen, coal mining, and paraffin products or residues of such substances.
PHYSICAL	
XXI - NOISE AND hearing damage	Mining, tunnel construction, quarrying (blasting, drilling), heavy engineering (cast iron, forging press), working with machines that run on powerful combustion engines, use of textile machinery, aircraft testing reactors.
XXII - VIBRATIONS (Disorders of muscles, tendons, bones, joints, peripheral blood vessels or peripheral nerves)	Metallurgical industry, shipbuilding and automotive, mining, agriculture (saws), pneumatic tools, vibrating tools, electric and manual driving trucks and buses.
XXIII - COMPRESSED AIR	work in coffins or pneumatic chambers and pneumatic caissons; operations with the use of diving; diving operations; Working with compressed air in pressurized tunnels.
XXIV - IONIZING RADIATION	extraction of radioactive minerals (treatment, purification, isolation and preparation for distribution), such as uranium; operation with nuclear reactors or neutron sources or other corpuscular radiation; work performed by exposure to X-ray, radio and radioactive substances for industrial, therapeutic and diagnostics; manufacturing and handling of radioactive chemicals and pharmaceuticals (uranium, radon,

	mesotório, thorium X, cesium 137 and others); manufacturing and application of luminescent products radíferos; researches and studies of X-rays and radioactive substances in laboratories.
BIOLOGICAL	
XXV - LIVE MICROORGANISMS AND INFECTIOUS PARASITES AND THEIR TOXIC	Agriculture, livestock, forestry, hunting (including trapping); veterinary; tannery.
Mycobacterium; virus hosted by arthropods; coccidióides; fungi; histoplasma; leptospira; Rickettsia; Bacillus (anthrax, tetanus); hookworm; trypanosome; pasteurella.	Construction, excavation of earth drains; irrigation canal; mining.
Hookworm; histoplasma; coccidióides; leptospira; bacillus; sepsis.	Handling and packaging of meat and fish.
Mycobacterium; brucellas; streptococci (erysipelas); fungus; Rickettsia; pasteurella.	Handling confined birds and birds.
Fungi; bacteria; mixovirus (Newcastle disease).	Working with hair, fur or wool.
Bacillus (anthrax) and pasteurella.	Veterinary.
Bacteria; mycobacteria, Brucella, fungi, leptospira, viruses, mixovirus; Rickettsia; pasteurella.	Hospital, laboratories and other environments involved in the treatment of diseases.
	Work in conditions of high temperature and humidity (kitchens, gymnasiums, swimming pools, etc.)..

Mycobacteria, viruses, other organisms responsible for diseases. Fungi (ringworm of the skin).	
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ORGANIC DUST

XXVI - COTTON, FLAX, HEMP, SISAL	Workers in various operations with dust from these products.
XXVII - physical, chemical or biological, that affect the skin, NOT FOUND IN OTHER ITEMS.	Workers most at risk: agricultural, construction in general, the chemical industry of eletro galvanoplastia; dyeing; of the plastic reinforced with fiberglass, paint, engineering services (cutting oil or lubricant); services health (drugs, anesthetics, disinfectants); treatment of livestock, the butcher.

Chair of Legal Affairs República Casa Civil Subchefia DECREE No. 6957 OF 9 SEPTEMBER 2009.

Amends the Social Security Regulation, approved by Decree 3048 of May 6, 1999, regarding the implementation, monitoring and evaluation of the Accident Prevention Factor - FAP.

THE PRESIDENT OF THE REPUBLIC, in exercise of the powers granted by art. 84, item IV of the Constitution, and in view of their Laws in 8212, to July 24, 1991, 8213, to July 24, 1991, and 11,430 of 26 December 2006

DECREES:

The first Art arts. 202-A, 303, 305 and 337 of the Social Security Regulation, approved by Decree 3048 of 6 May 1999, become effective with the following changes:

"Art 202-A.....

§ The first consists of a multiplier variable FAP in a continuous range of five tenths (0.5000) two integers (2.0000) applied to four decimal places, rounding on the criterion of the fourth decimal place, to be applied to their rate .

§ 2 For the purposes of the reduction or increase referred to in the chapeau, it will proceed to the discrimination performance of the company, within their own economic activity, from the creation of a composite index by the indices of severity, frequency and cost which balances their weight percentiles than fifty percent, thirty five percent and fifteen percent, respectively.

.....
§ 4.....

I - for the frequency index, the records of accidents and illnesses reported to Social Security through Communication Workers' Accident - CAT and accident benefits by nexus established by medical technicians INSS, even without CAT linked to them ;

II - to the severity index, all cases of sickness, accident allowance, disability retirement and pension, all of nature accident, which are assigned different weights to reflect the gravity of the occurrence, as follows:

a) pension for death: fifty weight percent;

b) disability retirement: weight thirty percent and

c) the sickness and accident allowance: ten percent weight to each, and

III - for the cost index, the values of rugged nature of the benefits paid or payable by Social Security, calculated as follows:

a) in cases of sickness, based on employee time off in months and fraction of months, and

b) in cases of death or disability, whether partial or total, by projection of the expected survival of the insured on the date of commencement of the benefit from the mortality tables constructed by the Brazilian Institute of Geography and Statistics - IBGE for the entire Brazilian population, considering only the national average for both sexes.

§ 5The Ministry of Social Security shall publish annually in the same month in the Official Gazette, the Heros of the percentiles of frequency, severity and cost per Division of the National Classification of Economic Activities - NCEA and disseminate the global network of computers each FAP company, with their national frequency, severity, cost and other elements that allow it to check their performance within their NCEA-Division.

.....
§ 7For annual calculation of FAP, we used the data from January to

December of each year to complete the two year period, from which the data from the initial year will be replaced by new annual data incorporated.

§ For the eighth after the company incorporated in January 2007, the FAP will be calculated from January 1 of the year following the year to complete two years of incorporation.

§ 9th Exceptionally, in the first processing of FAP used the data from April 2007 to December 2008.

§ 10. The methodology adopted by the National Social Security indicate the system of calculating and manner of implementation of criteria and indices accessories to the composition of the composite index of FAP.

"(NR)

"Art 303.....

§ 1

I - twenty-nine Boards of Appeals, with jurisdiction to prosecute in the first instance, appeals against the decisions handed down by the INSS regional bodies with regard to benefits administered by the municipality or in the determination of disputes concerning the FAP, which referred to in art. 202-A, according to the system to be defined in a joint act of the Ministries of Social Welfare and of Finance;

....." (NR)

"Section 305. The decisions of Social Security in the processes of interest of the beneficiaries of disputes concerning the determination of the FAP will fit resource for CRPS, as provided in these Rules and Bylaws of the Council.

....." (NR)

"Art 337.....

.....

§ 3 It is established the link between work and injury when technical epidemiological link is established between the company's activity and the inability motivating disease entity, listed in the International Classification of Diseases - ICD in accordance with the provisions in Schedule C of Annex II these Regulations.

....." (NR)

The second ArtAnnexes IIandV of the Social Security Regulationbecome effective as of the Attachments to this Decree.

Article 3 In 2010, the Accident Prevention Factor - FAP, in writing given by this Decree, shall be applied in more than one whole, with a reduction of twenty-five per cent, thus consisting of a variable multiplier in the range a full continuum of a whole and seventy-five cents.

4th Art This Decree shall enter into force upon its publication, and its effects on the new wording of Annex V of the Social Security Regulation, from the first day of January 2010, maintained until this date the contributions due to the law precedent.

Art is the fifth Repeals§ 3 of art. 202-A of the Social Security Regulation, approved by Decree 3048 of 6 May 1999.

Brasília, September 9, 2009, the 188th and 121st Independence of the Republic.

Luiz Inacio Lula da Silva

José Pimentel

ANNEX

"ANNEX II

PATHOGENS CAUSING OCCUPATIONAL DISEASES
OR WORK, AS PROVIDED IN ART. 20 OF LAW NO 8213 OF 1991

.....

LIST B

Note:

1. The diseases and their etiologic agents or risk factors of occupational nature listed are exemplary and complementary.

INFECTIOUS DISEASES RELATED WORK

(Group I of ICD-10)

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
I - Tuberculosis (A15-A19. -)	Occupational exposure to Mycobacterium tuberculosis (M. tuberculosis) or Mycobacterium bovis, in activities in biology labs, and activities performed by health personnel who provide direct contact with contaminated products or patients whose bacteriological tests are positive (Z57.8) (Table XXV) Hipersuscetibilidade worker exposed to dust from silica (sand-TB) (J65. -) Zoonosis caused by occupational exposure to Bacillus anthracis in activities that could put workers in direct contact with infected animals or carcasses of such animals; crafts or industrial fur, skin, leather or wool. (Z57.8) (Table XXV) Zoonosis caused by occupational exposure to Brucella melitensis, B. abortus, B. suis, B. kennels, etc., to
II - Anthrax (A22. -)	
III - Brucellosis (A23. -)	

DISEASES

Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE

work in slaughterhouses, packing plants, manipulation of meat products, dairy milking and manufacturing and related activities. (Z57.8) (Table XXV)

IV - Leptospirosis (A27. -)

Occupational exposure to *Leptospira icterohaemorrhagiae* (and other) species, exhibiting works in direct contact with dirty water or made in places likely to be soiled by animal waste carriers of germs; work carried out in mines, tunnels, sewers into local underground; work in watercourses; drainage works, contact with rodents; work with pets, and livestock; preparation of food of animal origin, fish, dairy, etc. .. (Z57.8) (Table XXV)

V - Tetanus (A35. -)

Exposure to *Clostridium tetani*, in circumstances of occupational accidents in agriculture, construction, industry, or commuting accidents (Z57.8) (Table XXV)

VI - psittacosis, ornithosis, Disease of poultry handlers (A70. -)

Zoonoses caused by occupational exposure to *Chlamydia psittaci* or *Chlamydia pneumoniae*, in studies on breeding birds or birds, Veterinary activities in zoos, and biological laboratories, etc.. (Z57.8) (Table XXV)

VII - Dengue fever [classical dengue] (A90. -)

Occupational exposure to mosquito (*Aedes aegypti*), vector of dengue arboviruses, especially in activities in endemic areas in public health services, and jobs in research laboratories, among others. (Z57.8) (Table XXV)

VIII - Yellow Fever (A95. -)

Occupational exposure to mosquito (*Aedes aegypti*), vector of arboviruses of the Yellow Fever mainly on

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
IX - Viral Hepatitis (B15-B19. -)	<p>activities in endemic areas, work in public health, and work of research laboratories, among others. (Z57.8) (Table XXV)</p> <p>Occupational exposure to Hepatitis A virus (HAV), Hepatitis B Virus (HBV), Hepatitis C Virus (HCV), Hepatitis D (HDV), Hepatitis E virus (HEV) in work involving handling, packaging or use of human blood or its derivatives; work with "sewage" and sewers; work in contact with material from patients or objects contaminated by them. (Z57.8) (Table XXV)</p>
<p>X - Disease Human Immunodeficiency Virus (HIV) (B20-B24. -)</p>	<p>Occupational exposure to human immuno-deficiency (HIV), especially among healthcare workers, due to cutting / piercing injuries from needles or contaminated surgical material, and the handling, packaging or use of blood or blood products, and contact material from infected patients. (Z57.8) (Table XXV)</p>
<p>XI - Ringworm (B35. -) and Other Superficial Mycoses (B36. -)</p>	<p>Occupational exposure to fungi of the genera Epidermophyton, Microsporum and Trichophyton in work under conditions of high temperature and humidity (kitchens, gymnasiums, swimming pools) and other situations of occupational exposure. (Z57.8) (Table XXV)</p>
XII - Candidiasis (B37. -)	<p>Occupational exposure to <i>Candida albicans</i>, <i>Candida glabrata</i>, etc.. in jobs that require long immersion of hands in water and mechanical irritation of the hands, such as cleaning workers, laundresses, cooks, among others. (Z57.8) (Table XXV)</p>

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
XIII - Paracoccidioidomycosis (South American blastomycosis, Brazilian blastomycosis, Lutz's disease) (B41. -)	Occupational exposure to <i>P. brasiliensis</i> , mainly for agricultural and forestry and in endemic areas. (Z57.8) (Table XXV)
XIV - Malaria (B50 - B54 .-)	Occupational exposure to <i>Plasmodium malariae</i> , <i>Plasmodium vivax</i> , <i>Plasmodium falciparum</i> and other protozoa, mainly in mining, building dams or highways, in oil extraction and other activities that require the entry of workers in endemic areas (Z57.8) (Table XXV)
XV - Cutaneous leishmaniasis (B55.1) or mucocutaneous leishmaniasis (B55.2)	Occupational exposure to <i>Leishmania braziliensis</i> , mainly for agricultural and forestry and in endemic areas, and other situations of occupational exposure. (Z57.8) (Table XXV)

**Neoplasm (tumor) WORK-RELATED
(GROUP II of ICD-10)**

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
I - malignant neoplasm of the stomach (C16. -)	Asbestos or asbestos (X49. -; Z57.2) (Table II) 1. Arsenic and arsenic compounds (X48. -; X49. -, Z57.5) (Table I)
II - angiosarcoma of the liver (C22.3)	2. Vinyl Chloride (X46. -; Z57.5) (Table XIII) 1. Vinyl Chloride (X46. -; Z57.5) (Table XIII)
III - malignant neoplasm of the pancreas (C25. -)	2. Epichlorohydrin (X49. -; Z57.5) 3. Alifáticos and aromatic hydrocarbons in the Petroleum Industry (X46. -; Z57.5)
IV - malignant neoplasm of the nasal cavity and paranasal sinuses (C30-C31. -)	1. Ionizing radiation (W88. -; Z57.1) (Table XXIV) 2. Nickel and compounds (X49. -; Z57.5) 3. Dusts from wood and other organic

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
V - malignant neoplasm of the larynx (C32. -)	<p>dusts the furniture industry (X49. -; Z57.2)</p> <p>4. Dusts of the leather industry (X49. -; Z57.2)</p> <p>5. Organic dust (in the textile industry and in bakeries) (X49. -; Z57.2)</p> <p>6. Petroleum industry (X46. -; Z57.5)</p>
VI - malignant neoplasm of bronchus and lung (C34. -)	<p>Asbestos or asbestos (Z57.2) (Table II)</p> <p>1. Arsenic and arsenic compounds (X48. -; X49. -, Z57.4 and Z57.5) (Table I)</p> <p>2. Or Asbestos Asbestos (X49. -; Z57.2) (Table II)</p> <p>3. Beryllium (X49. -; Z57.5) (Table IV)</p> <p>4. Cadmium or its compounds (X49. -; Z57.5) (Table VI)</p> <p>5. Chromium and its toxic compounds (X49. -; Z57.5) (Table X)</p> <p>6. Vinyl Chloride (X46. -; Z57.5) (Table XIII)</p> <p>7. Chloromethyl ethers (X49. -; Z57.5) (Table XIII)</p> <p>8. Silica-free (Z57.2) (Table XVIII)</p> <p>9. Tar, pitch, bitumen, coal mining, paraffin and waste products such substance (X49. -; Z57.5) (Table XX)</p> <p>10. Ionizing radiation (W88. -; Z57.1) (Table XXIV)</p> <p>11. Emissions from coke ovens (X49. -; Z57.5)</p> <p>12. Nickel and compounds (X49. -; Z57.5)</p> <p>13. Acrylonitrile (X49. -; Z57.5)</p> <p>14. Aluminum industry (foundries) (X49. -; Z57.5)</p> <p>15. Mist of mineral oil (cutting oil) (X49. -; Z57.5)</p> <p>16. Metal foundries (X49. -; Z57.5)</p>

DISEASES

**Etiologic agent or RISK FACTORS
OF OCCUPATIONAL NATURE**

VII - malignant neoplasm of bone and articular cartilage of limbs (Includes "Bone Sarcoma") (C40. -)

Ionizing radiation (W88. -; Z57.1) (Table XXIV)

1. Arsenic and arsenic compounds (X49. -; Z57.4 and Z57.5) (Table I)

2. Tar, pitch, bitumen, mineral coal, paraffin and waste products of these substances cause skin epitheliomas (X49. -; Z57.5) (Table XX)

VIII - Other malignant neoplasms of skin (C44. -)

3. Ionizing radiation (W88. -; Z57.1) (Table XXIV)

4. Ultraviolet radiation (W89; Z57.1)

IX - Mesothelioma (C45. -):
Mesothelioma of pleura (C45.0), mesothelioma of the peritoneum (C45.1) and mesothelioma of the pericardium (C45.2)

Asbestos or asbestos (X49. -; Z57.2) (Table II)

1. Tar, pitch, bitumen, mineral coal, paraffin and waste products such substance (X49. -; Z57.5) (Table XX)

2. Aromatic amines and their derivatives (Beta-naphthylamine and 2-chloroaniline, benzidine, o-toluidine, 4-chloro-ortho-toluidine (X49. -; Z57.5)

X - malignant neoplasm of the bladder (C67. -)

3. Emissions from coke ovens (X49. -; Z57.5)

1. Benzene (X46. -; Z57.5) (Table III)

2. Ionizing radiation (W88. -; Z57.1) (Table XXIV)

3. Ethylene oxide (X49. -; Z57.5)

XI - Leukemia (C91-C95. -)

4. Antineoplastic agents (X49. -; Z57.5)

5. Electromagnetic fields (W90. -; Z57.5)

6. Chlorinated Pesticides (chlordane and heptachlor) (X48. -; Z57.4)

DISEASES OF BLOOD AND HEMATOPOIETIC ORGANS

RELATED WORK (Group III of ICD-10)

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
I - Myelodysplastic Syndromes (D46. -)	1. Benzene (X46. -; Z57.5) (Table III) 2. Ionizing radiation (W88. -; Z57.1) (Table XXIV)
II - Other anemias due to enzyme disorders (D55.8)	Lead or its toxic compounds (X49. -; Z57.5) (Table VIII)
III - hemolytic anemia syndrome (D59.2)	Nitro and amino derivatives of benzene (X46. -; Z57.5)
IV - Aplastic due to other external agents (D61.2)	1. Benzene (X46. -; Z57.5) (Table III) 2. Ionizing radiation (W88. -) (Table XXIV)
V - Aplastic Anemia unspecified NOS hypoplastic anemia, bone marrow hypoplasia (D61.9)	1. Benzene (X46. -; Z57.5) (Table III) 2. Ionizing radiation (W88. -; Z57.1) (Table XXIV)
VI - Sideroblastic Anemia secondary to toxins (including "Anemia Hypochromic, microcytic, with Reticulocytosis") (D64.2)	Lead or its toxic compounds (X46. -; Z57.5) (Table VIII)
VII - Purpura and other hemorrhagic manifestations (D69. -)	1. Benzene (X46. -; Z57.5) (Table III) 2. Vinyl Chloride (X46. -) (Table XIII) 3. Ionizing radiation (W88. -; Z57.1) (Table XXIV)
VIII - agranulocytosis (Neutropenia toxic) (D70)	1. Benzene (X46. -; Z57.5) (Table III) 2. Ionizing radiation (W88. -; Z57.1) (Table XXIV) 3. Derivatives of Phenol, Pentachlorophenol, Hydroxybenzonitrile (X49. -; XZ57.5)
IX - Other specified disorders of white blood cells, leukocytosis, leukemoid reaction (D72.8)	1. Benzene (X46. -; Z57.5) (Table III) 2. Ionizing radiation (W88. -; Z57.1) (Table XXIV)
X - Methemoglobinemia (D74. -)	Aromatic amines and their derivatives (X49. -; Z57.5)

DISEASES ENDOCRINE, NUTRITIONAL AND METABOLIC

RELATED WORK (Group IV ICD-10)

DISEASES

**Etiologic agent or RISK FACTORS
OF OCCUPATIONAL NATURE**

1. Lead or its toxic compounds (X49. -; Z57.5) (Table VIII)
 2. Halogenated hydrocarbons (chlorobenzene and its derivatives) (X46. -; Z57.5) (Table XIII)
 3. Tiuracil (X49. -; Z57.5)
 4. Tiocinatos (X49. -; Z57.5)
 5. Thiourea (X49. -; Z57.5)
 - Chlorobenzene and its derivatives (X46. -; Z57.4 and Z57.5) (Table XIII)
- I - Hypothyroidism due to exogenous substances (E03. -)
- II - Other Porphyrrias (E.80.2)

MENTAL DISORDERS AND BEHAVIOR

WORK-RELATED (Group V of ICD-10)

DISEASES

**Etiologic agent or RISK FACTORS OF
OCCUPATIONAL NATURE**

1. Manganese X49. -, Z57.5) (Table XV)
 2. Asphyxiating substances: CO, H2S, etc.. (Sequel) (X47. -; Z57.5) (Table XVII)
 3. Sulfide Carbon (X49. -; Z57.5) (Table XIX)
 1. Methyl Bromide (X46. -; Z57.4 and Z57.5) (Table XIII)
 2. Sulfide Carbon (X49. -; Z57.5) (Table XIX)
 1. Toluene and other aromatic solvents neurotoxic (X46. -; Z57.5) (Table III)
 2. Lead or its toxic compounds (X49. -; Z57.5) (Table VIII)
 3. Trichloroethylene, Tetrachloroethylene, trichloroethane and other halogenated organic solvents neurotoxicity (X46. -; Z57.5) (Table XIII)
 4. Methyl Bromide (X46. -; Z57.4 and Z57.5) (Table XIII)
 5. Manganese and its toxic compounds (X49. -; Z57.5) (Table XV)
 6. Mercury and its toxic compounds (X49. -; Z57.4 and Z57.5) (Table XVI)
- I - Dementia in other diseases classified elsewhere (F02.8)
- II - Delirium, not superimposed on dementia, as described (F05.0)
- III - Other mental disorders due to brain damage and dysfunction and physical illness (F06. -): Mild Cognitive Impairment (F06.7)

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|---|---|
| <p>IV - Personality disorders and behavioral disorders due to illness, injury and dysfunction in personality (F07. -): Disorder Organic Personality (F07.0) Other disorders of personality and behavior due to illness, injury or brain dysfunction (F07.8)</p> | <p>7. Sulfide Carbon (X49. -; Z57.5) (Table XIX)</p> |
| | <p>8. Neurotoxic Other organic solvents (X46. -; X49. -, Z57.5)</p> |
| | <p>1. Toluene and other aromatic solvents neurotoxic (X46. -; Z57.5) (Table III)</p> |
| | <p>2. Trichloroethylene, Tetrachloroethylene, trichloroethane and other halogenated organic solvents neurotoxicity (X46. -; Z57.5) (Table XIII)</p> |
| | <p>3. Methyl Bromide (X46. -; Z57.4 and Z57.5) (Table XIII)</p> |
| | <p>4. Manganese and its toxic compounds (X49. -; Z57.5) (Table XV)</p> |
| | <p>5. Mercury and its toxic compounds (X49. -; Z57.4 and Z57.5) (Table XVI)</p> |
| <p>V - organic or symptomatic mental disorder unspecified (F09. -)</p> | <p>6. Sulfide Carbon (X49. -; Z57.5) (Table XIX)</p> |
| | <p>7. Neurotoxic Other organic solvents (X46. -; X49. -, Z57.5)</p> |
| | <p>1. Toluene and other aromatic solvents neurotoxic (X46. -; Z57.5) (Table III)</p> |
| | <p>2. Trichloroethylene, Tetrachloroethylene, trichloroethane and other halogenated organic solvents neurotoxicity (X46. -; Z57.5) (Table XIII)</p> |
| | <p>3. Methyl Bromide (X46. -; Z57.5) (Table XIII)</p> |
| | <p>4. Manganese and its toxic compounds (X49. -; Z57.5) (Table XV)</p> |
| | <p>5. Mercury and its toxic compounds (X49. -; Z57.4 and Z57.5) (Table XVI)</p> |
| <p>VI - Mental and behavioral disorders due to alcohol abuse: Chronic Alcoholism (Related Work) (F10.2)</p> | <p>6. Sulfide Carbon (X49. -; Z57.5) (Table XIX)</p> |
| | <p>7. Neurotoxic Other organic solvents (X46. -; X49. -, Z57.5)</p> |
| | <p>1. Problems related to employment and unemployment, difficult working conditions (Z56.5)</p> |
| | <p>2. Condition relating to working conditions (Y96)</p> |

1. Toluene and other aromatic solvents neurotoxic (X46. -; Z57.5) (Table III)
 2. Trichloroethylene, Tetrachloroethylene, trichloroethane and other halogenated organic solvents neurotoxicity (X46. -; Z57.5) (Table XIII)
 3. Methyl Bromide (X46. -; Z57.4 and Z57.5) (Table XIII)
- VII - Depressive Episodes (F32. -)
4. Manganese and its toxic compounds (X49. -; Z57.5) (Table XV)
 5. Mercury and its toxic compounds (X49. -; Z57.4 and Z57.5) (Table XVI)
 6. Sulfide Carbon (X49. -; Z57.5) (Table XIX)
 7. Neurotoxic Other organic solvents (X46. -; X49. -, Z57.5)
- VIII - Reactions to Stress Severe and adjustment disorders (F43. -): State of Stress Post-Traumatic Stress Disorder (F43.1)
1. Other physical and mental difficulties related to work: reaction after work accident serious or catastrophic assault on or after work (Z56.6)
 2. Condition relating to working conditions (Y96)
1. Toluene and other aromatic solvents neurotoxic (X46. -; Z57.5) (Table III)
 2. Trichloroethylene, Tetrachloroethylene, trichloroethane and other halogenated organic solvents (X46. -; Z57.5) (Table XIII)
 3. Methyl Bromide (X46. -; Z57.4 and Z57.5) (Table XIII)
 4. Manganese and its toxic compounds (X49. -; Z57.5) (Table XV)
 5. Mercury and its toxic compounds (X49. -; Z57.4 and Z57.5) (Table XVI)
 6. Sulfide Carbon (X49. -; Z57.5) (Table XIX)
 7. Neurotoxic Other organic solvents (X46. -; X49. -, Z57.5)
- IX - Neurasthenia (Includes "Fatigue Syndrome") (F48.0)
- X - Other specified neurotic disorders (including "Neurosis Professional") (F48.8)
- Problems related to employment and unemployment (Z56. -): Unemployment (Z56.0); Changing jobs (Z56.1); Threat of

	job loss (Z56.2); Pace drudgery (Z56.3) Disagreement with boss and coworkers (difficult working conditions) (Z56.5) Other physical and mental difficulties related to work (Z56.6)
XI - Disorder of sleep-wake cycle due to factors Non-Organic (F51.2 Nonorganic)	1. Problems related to Employment and unemployment: Poor adaptation to the organization of working hours (or Shift Work Night Work) (Z56.6) 2. Condition relating to working conditions (Y96)
XII - Feeling of Being Finished ("Burn-Out Syndrome", "Professional Burnout Syndrome") (Z73.0)	1. Pattern of drudgery (Z56.3) 2. Other difficulties related to physical and mental work (Z56.6)

NERVOUS SYSTEM DISEASES RELATED WORK (Group VI of the ICD-10)

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
I - cerebellar ataxia (G11.1)	Mercury and its toxic compounds (X49. - ; Z57.4 and Z57.5) (Table XVI)
II - Secondary parkinsonism due to other external agents (G21.2)	Manganese and its toxic compounds (X49. -; Z57.5) (Table XV) 1. Methyl bromide (X46. -; Z57.4 and Z57.5) (Table XIII) 2. Tetrachloroethane (X46. -; Z57.5) (Table XIII)
III - Other specified forms of tremor (G25.2)	3. Mercury and its toxic compounds (X49. -; Z57.4 and Z57.5) (Table XVI) 4. Neurotoxic Other organic solvents (X46. -; X49 .-, Z57.5) 1. Mercury and its toxic compounds (X49. -; Z57.4 and Z57.5) (Table XVI) 2. Methylene chloride (dichloromethane) and other halogenated solvents neurotoxic (X46. -; Z57.5) (Table XIII)
IV - extrapyramidal movement disorder unspecified (G25.9)	Problems related to employment and unemployment: Poor adaptation to the organization of working hours (or Shift Work Night Work) (Z56.6)
V - Disorders of the sleep-wake cycle (G47.2)	Trichloroethylene and other solvents
VI - Disorders of the trigeminal	

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
nerve (G50. -)	neurotoxic halogenated (X46. -; Z57.5) (Table XIII)
VII - Disorders of olfactory nerve (G52.0) (Includes "Anosmia")	1. Cadmium or its compounds (X49. -; Z57.5) (Table VI) 2. Hydrogen sulfide (X49. -; Z57.5) (Table XVII)
VIII, Disorders of the brachial plexus (Thoracic Outlet Syndrome, Thoracic Outlet Syndrome) (G54.0)	Awkward positions and repetitive movements (Z57.8)
IX - Mononeuropathies Member Institutions (G56. -): Carpal tunnel syndrome (G56.0) Other Injuries of the Median Nerve: pronator teres syndrome (G56.1); Canal Guyon syndrome (G56.2); Injury of the ulnar nerve (ulnar): cubital tunnel syndrome (G56.2) Lesion of the Radial Nerve (G56.3) Other Mononeuropathies of Upper Limbs: Compression of the suprascapular nerve (G56.8)	Awkward positions and repetitive movements (Z57.8)
X - Mononeuropathies lower limb (G57. -): Lateral popliteal nerve injury (G57.3)	Awkward positions and repetitive movements (Z57.8) 1. Arsenic and arsenic compounds (X49. -; Z57.4 and Z57.5) (Table I) 2. Lead and its toxic compounds (X49. -; Z57.5) (Table VIII) 3. Phosphorus (X48. -; X49. -, Z57.4 and Z57.5) (Table XII) 4. Sulfide Carbon (X49. -; Z57.5) (Table XIX) 5. N-Hexane (X46. -; Z57.5) (Table XIII) 6. Methyl-n-Butyl Ketone (MBK) (X46. -; Z57.5)
XI - Polyneuropathy due to other toxic agents (G62.2)	Ionizing radiation (X88. -; Z57.1) (Table XXIV)
XII - Radiation-induced polyneuropathy (G62.8)	1. Arsenic and arsenic compounds (X49.
XIII - Acute Toxic Encephalopathy	

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
(G92.1)	-; Z57.4 and Z57.5) (Table I) 2. Lead and its toxic compounds (X49. -; Z57.5) (Table VIII) 3. Aliphatic or aromatic hydrocarbons (halogenated its neurotoxic) (X46. -; Z57.5) (Table XIII) 4. Mercury and its toxic derivatives (X49. -; Z57.4 and Z57.5) (Table XVI) 1. Toluene and Xylene (X46. -; Z57.5) (Table III) 2. Lead and its toxic compounds (X49. -; Z57.5) (Table VIII) 3. Neurotoxic halogenated organic solvents (X46. -; Z57.5) (Table XIII) 4. Mercury and its toxic compounds (X49. -; Z57.5) (Table XVI) 5. Asphyxiating substances: CO, H ₂ S, etc.. (Sequel) (X47. -; Z57.5) (Table XVII) 6. Sulfide Carbon (X49. -; Z57.5) (Table XIX)
XIV - Chronic Toxic Encephalopathy (G92.2)	

**DISEASES OF THE EYE AND NOTES RELATED WORK
(Group VII of ICD-10)**

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
I - Blepharitis (H01.0)	1. Arsenic and arsenic compounds (X49. -; Z57.4 and Z57.5) (Table I) 2. Ionising Radiation (W88. -; Z57.1) (Table XXIV) 3. Cement (X49. -; Z57.2) 1. Arsenic and arsenic compounds (X49. -; Z57.4 and Z57.5) (Table I) 2. Beryllium and its toxic compounds (X49. -; Z57.5) (Table IV) 3. Fluorine and toxic compounds (X49. -) (Table XI) 4. Iodine (X49. -; Z57.5) (Table XIV) 5. Ethyl Chloride (X46. -; Z57.5) (Table XIII)
II - Conjunctivitis (H10)	

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
III - Keratitis and keratoconjunctivitis (H16)	6. Carbon tetrachloride (X46. -; Z57.5) (Table XIII)
	7. Other toxic halogenated solvents (X46. -; Z57.4 and Z57.5) (Table XIII)
	8. Sulfide acid (hydrogen sulfide) (X49. -; Z57.5) (Table XVII)
	9. Ionizing radiation (W88. -; Z57.1) (Table XXIV)
	10. Ultraviolet Radiation (W89; Z57.1)
	11. Acrylates (X49. -; Z57.5)
	12. Cement (X49. -; Z57.2)
	13. Enzymes of animal, plant or bacterial (X44. -; Z57.2)
	14. Furfural and furfuryl alcohol (X45. -; Z57.5)
	15. Organic Isocyanates (X49. -; Z57.5)
	16. Selenium and its compounds (X49. -; Z57.5)
	1. Arsenic and arsenic compounds (X49. -; Z57.4 and Z57.5) (Table I)
	2. Sulfide acid (hydrogen sulfide) (X49. -; Z57.5) (Table XVII)
	3. Ionizing radiation (W88. -; Z57.1) (Table XXIV)
	4. Infrared Radiation (W90. -; Z57.1)
	5. Ultraviolet Radiation (W89. -; Z57.1)
IV - Cataract (H28)	1. Ionizing radiation (W88. -; Z57.1) (Table XXIV)
	2. Infrared Radiation (W90. -; Z57.1)
V - Chorioretinal Inflammation (H30)	Manganese and its toxic compounds (X49. -; Z57.5) (Table XV)
	1. Methyl bromide (X46. -; Z57.4 and Z57.5) (Table XIII)
	2. Methylene chloride (dichloromethane) and other chlorinated solvents neurotoxic (X46. -; Z57.5) (Table XIII)
VI - Optic neuritis (H46)	3. Carbon tetrachloride (X46. -; Z57.5) (Table XIII)

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
	4. Sulfide Carbon (X49. -; Z57.5) (Table XIX)
	5. Methanol (X45. -; Z57.5)
	1. Methyl bromide (X46. -; Z57.4 and Z57.5) (Table XIII)
VII-subjective visual disturbances (H53. -)	2. Methylene chloride and other chlorinated solvents neurotoxic (X46. -; Z57.5) (Table XIII)

**DISEASES OF THE EAR RELATED WORK
(Group VIII of the ICD-10)**

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
I - non-suppurative otitis media (H65.9)	1. "Compressed Air" (W94. -; Z57.8) (Table XXIII) 2. Atmospheric pressure lower than the standard pressure (W94. -; Z57.8)
II-perforation of the tympanic membrane (or H72 S09.2)	1. "Compressed Air" (W94. -; Z57.8) (Table XXIII) 2. Atmospheric pressure lower than the standard pressure (W94. -; Z57.8)
III - Other peripheral vertigo (H81.3)	Methylene chloride and other toxic halogenated solvents (X46. -; Z57.5) (Table XIII) 1. Methyl bromide (X46. -; Z57.4 and Z57.5) (Table XIII)
IV - Labyrinthitis (H83.0)	2. "Compressed Air" (W94. -; Z57.8) (Table XXIII)
V - Effects of noise on the inner ear / Hearing Loss Caused by Noise and Acoustic Trauma (H83.3)	Occupational exposure to noise (Z57.0; W42. -) (Table XXI) 1. Otoneurotóxicos Homologs of Benzene (Toluene and Xylene) (X46. -; Z57.5) (Table III)
VI - Hearing loss ototoxic (H91.0)	2. Otoneurotóxicos organic solvents (X46. -; Z57.8) (Table XIII)
VII - Hearing Ear pain and Secretion (H92. -): otalgia (H92.0), otorrhea (H92.1) or otorrhea	"Air Power" (W94. -; Z57.8) (Table XXIII)

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
(H92.2) VIII - Other abnormal auditory perceptions: Amendment of Temporary Auditory Threshold, Impairment of Auditory Discrimination and Hyperacusis (H93.2)	Occupational exposure to noise (Z57.0; X42 .-) (Table XXI)
IX - Other specified disorders of ear (H93.8)	1. Methyl bromide (X46. -; Z57.4 and Z57.5) (Table XIII) 2. "Compressed Air" (W94. -; Z57.8) (Table XXIII) 1. "Compressed Air" (W94. -; Z57.8) (Table XXIII)
X - Ear barotrauma (T70.0)	2. Changes in atmospheric pressure or water pressure on the environment (W94. -; Z57.8) 1. "Compressed Air" (W94. -; Z57.8) (Table XXIII)
XI - Sinus barotrauma (T70.1)	2. Changes in atmospheric pressure or water pressure on the environment (W94. -) 1. "Compressed Air" (W94. -; Z57.8) (Table XXIII)
XII - "Coffins of Evil" (Sickness Decompression) (T70.4)	2. Changes in atmospheric pressure or water pressure on the environment (W94. -; Z57.8) 1. "Compressed Air" (W94. -; Z57.8) (Table XXIII)
XIII - Syndrome due to displacement of air from an explosion (T70.8)	2. Changes in atmospheric pressure or water pressure on the environment (W94. -; Z57.8)

CIRCULATORY SYSTEM DISEASES RELATED WORK

(Group IX of the ICD-10)

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
I - Hypertension (I10. -)	1. Lead or its toxic compounds (X49. -; Z57.5) (Table VIII) 2. Occupational Exposure to Noise (Z57.0; X42 .-) (Table XXI)

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
	3. Problems related to employment and unemployment (Z56. -)
	1. Carbon Monoxide (X47. -; Z57.5) (Table XVII)
	2. Sulfide Carbon (X49. -; Z57.5) (Table XIX)
II - Angina Pectoris (I20. -)	3. Nitroglycerin and other nitric acid esters (X49. -; Z57.5)
	4. Problems related to employment and unemployment (Z56. -)
	1. Carbon Monoxide (X47. -; Z57.5) (Table XVII)
	2. Sulfide Carbon (X49. -; Z57.5) (Table XIX)
III - Acute Myocardial Infarction (I21. -)	3. Nitroglycerin and other nitric acid esters (X49. -; Z57.5)
	4. Problems related to employment and unemployment (Z56. -)
IV - Cor Pulmonale or SOE Disease Cardio Pulmonary Disease (I27.9)	Complication of evolutionary severe pneumoconiosis, silicosis primarily (Z57.2) (Table XVIII)
V - boards epicardial or pericardial (I34.8)	Asbestos or asbestos (W83. -; Z57.2) (Table II)
	1. Halogenated aliphatic hydrocarbons (X46. -) (Table XIII)
	2. Carbon Monoxide (X47. -; Z57.5) (Table XVII)
VI - Cardiac Arrest (I46. -)	3. Other agents potentially causing cardiac arrhythmia (Z57.5)
	1. Arsenic and arsenic compounds (X49. -; Z57.5) (Table I)
	2. Lead or its toxic compounds (X49. -; Z57.5) (Table VIII)
	3. Halogenated aliphatic hydrocarbons (X46. -; Z57.5) (Table XIII)
VII - Cardiac arrhythmias (I49. -)	4. Mercury and its toxic compounds (X49. -; Z57.5) (Table XVI)
	5. Carbon Monoxide (X47. -; Z57.5) (Table XVII)

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
	6. Agrochemicals organophosphates and carbamates (X48; Z57.4) (Tables XII and XXVII)
	7. Occupational exposure to cobalt (X49. -; Z57.5)
	8. Nitroglycerin and other nitric acid esters (X49. -; Z57.5)
	9. Problems related to employment and unemployment (Z56. -)
VIII - Atherosclerosis (I70. -) and Disease Angioplasty (I25.1)	Carbon sulfide (X49. -; Z57.5) (Table XIX)
	1. Vinyl chloride (X46. -; Z57.5) (Table XIII)
IX - Raynaud's syndrome (I73.0)	2. Vibrations localized (W43. -; Z57.7) (Table XXII)
	3. Working at low temperatures (cold) (W93. -; Z57.6)
	1. Vinyl chloride (X46. -; Z57.5) (Table XIII)
X - Acrocyanosis and acroparesthesias (I73.8)	2. Vibrations localized (W43. -; Z57.7) (Table XXII)
	3. Working at low temperatures (cold) (W93. -; Z57.6)

DISEASES OF THE RESPIRATORY SYSTEM RELATED WORK (Group X of the ICD-10)

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
I - Acute pharyngitis, unspecified ("acute angina", "sore throat") (J02.9)	1. Bromine (X49. -; Z57.5) (Table V)
	2. Iodine (X49. -; Z57.5) (Table XIV)
II - Acute laryngotracheitis (J04.2)	1. Bromine (X49. -; Z57.5) (Table V)
	2. Iodine (X49. -; Z57.5) (Table XIV)
	1. Sintered metal carbides tungsten (X49. -; Z57.2 and Z57.5) (Table VII)
	2. Chromium and its toxic compounds (X49. -; Z57.5) (Table X)
III - Other Allergic rhinitis (J30.3)	3. Dust from cotton, flax, hemp or sisal (Z57.2) (Table XXVI)
	4. Acrylates (X49. -; Z57.5)

DISEASES

**Etiologic agent or RISK FACTORS
OF OCCUPATIONAL NATURE**

5. Formaldehyde and its polymers (X49. -; Z57.5)
 6. Aromatic amines and their derivatives (X49. -; Z57.5)
 7. Phthalic anhydride (X49. -; Z57.5)
 8. Azodicarbonamide (X49. -; Z57.5)
 9. Carbides of heavy metals: cobalt and titanium (Z57.2)
 10. Enzymes of animal, plant or bacterial (X44. -; Z57.3)
 11. Furfural and furfuryl alcohol (X45. -; Z57.5)
 12. Organic Isocyanates (X49. -; Z57.5)
 13. Nickel and compounds (X49. -; Z57.5)
 14. Pentoxide vanadium (X49. -; Z57.5)
 15. Products of pyrolysis of plastics, vinyl chloride, Teflon (X49. -; Z57.5)
 16. Sulfites, bisulfite and persulphates (X49. -; Z57.5)
 17. Medicines: macrolides; ranetidina; penicillin and its salts; cephalosporins (X44. -; Z57.3)
 18. Aerosols in animal protein (Z57.3)
 19. Other substances of plant origin (cereals, flour, sawdust, etc.). (Z57.2)
 20. Susbtâncias Other chemical sensitizers and respiratory (X49. -; Z57.2) (Table XXVII)
 1. Arsenic and arsenic compounds (X49. -; Z57.4 and Z57.5) (Table I)
 2. Chlorine gas (X47. -; Z57.5) (Table IX)
 3. Chromium and its toxic compounds (X49. -) (Table X)
 4. Gas Hydrogen fluoride and fluorine (X47. -; Z57.5) (Table XI)
 5. Ammonia (X47. -; Z57.5)
 6. Sulfur dioxide (X49. -; Z57.5)
- IV - Chronic rhinitis (J31.0)

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
V - Chronic pharyngitis (J31.2)	7. Cement (Z57.2) 8. Phenol and homologues (X46. -; Z57.5) 9. Mists of mineral acids (X47. -; Z57.5) 10. Nickel and compounds (X49. -; Z57.5) 11. Selenium and its compounds (X49. -; Z57.5) Bromine (X49. -; Z57.5) (Table V)
VI - Chronic Sinusitis (J32. -)	1. Bromine (X49. -; Z57.5) (Table V) 2. Iodine (X49. -; Z57.5) (Table XIV) 1. Arsenic and arsenic compounds (X49. -; Z57.4 and Z57.5) (Table I) 2. Cadmium or its compounds (X49. -; Z57.5) (Table VI)
VII - Ulceration or Necrosis of Nasal Septum (J34.0)	3. Chromium and its toxic compounds (X49. -; Z57.5) (Table X) 4. Aeoressóis Solutions and hydrocyanic acid and its derivatives (X47. -; Z57.5) (Table XVII)
VIII - Perforation of Nasal Septum (J34.8)	1. Arsenic and arsenic compounds (X49. -; Z57.4 and Z57.5) (Table I) 2. Chromium and its toxic compounds (X49. -; Z57.5) (Table X)
IX - Chronic laryngotracheitis (J37.1)	Bromine (X49. -; Z57.5) (Table V) 1. Chlorine gas (X47. -; Z57.5) (Table IX)
X - Other chronic obstructive pulmonary diseases (Includes: "Asthma Obstructive", "Chronic Bronchitis", "asthmatic bronchitis", "Bronchitis Chronic Obstructive") (J44. -)	2. Occupational exposure to silica dust free (Z57.2-) (Table XVIII) 3. Occupational exposure to cotton dust, flax, hemp or sisal (Z57.2) (Table XXVI) 4. Ammonia (X49. -; Z57.5) 5. Sulfur dioxide (X49. -; Z57.5) 6. Mists and aerosols of mineral acids (X47. -; Z57.5) 7. Occupational exposure to coal dust (Z57.2)
XI - Asthma (J45. -)	Same list of sensitizing substances that

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
	produce allergic rhinitis (X49. -; Z57.2, Z57.4 and Z57.5)
XII - the Pneumoconiosis Coal Workers (J60. -)	1. Occupational exposure to dust coal (Z57.2) 2. Occupational exposure to silica dust-free (Z57.2) (Table XVIII)
XIII - Pneumoconiosis due to asbestos (asbestosis) and other mineral fibers (J61. -)	Occupational exposure to asbestos or asbestos dust (Z57.2) (Table II)
XIV - Pneumoconiosis due to silica dust (silicosis) (J62.8)	Occupational exposure to silica dust-free (Z57.2) (Table XVIII)
XV - berylliosis (J63.2)	Occupational exposure to beryllium dust and toxic compounds (Z57.2) (Table IV)
XVI - Siderosis (J63.4)	Occupational exposure to iron dust (Z57.2)
XVII - Estanhose (J63.5)	Occupational exposure to dust from tin (Z57.2) 1. Occupational exposure to dust from tungsten carbide (Z57.2) (Table VII) 2. Occupational exposure to dust from hard metal carbide (cobalt, titanium, etc..) (Z57.2)
XVIII - Pneumoconiosis due to other inorganic dust unspecified (J63.8)	3. Occupational exposure to phosphate rock (Z57.2) 4. Occupational exposure to dust from alumina (Al ₂ O ₃) ("Shaver's disease") (Z57.2)
XIX - Pneumoconiosis associated with tuberculosis (silico-tuberculosis ") (J65. -)	Occupational exposure to silica dust-free (Z57.2) (Table XVIII)
XX - Diseases of the airways due to organic dust (J66. -): Byssinosis (J66.0), other organic dusts due to unspecified (J66.8)	Occupational exposure to cotton dust, flax, hemp, sisal (Z57.2) (Table XXVI)
XXI - Hypersensitivity Pneumonitis 1. Organic Dust (J67. -): the Lung Farmer (Farmer's Lung or) (J67.0); Bagaçose (J67.1), Bird Fancier's	1. Occupational exposure to dust containing microorganisms and parasites live infectious and toxic products (Z57.2) (Table XXV)

DISEASES

Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE

Lung (J67.2); Suberose (J67.3) Malt2. Occupational exposure to other Workers' Lung (J67.4) Lung those who work with Mushrooms (J67.5)

Pulmonary Disease Due to Air Conditioning Systems and Air Humidification (J67.7)

Hypersensitivity pneumonitis due to other organic dusts (J67.8)

Hypersensitivity Pneumonitis Due to Dust Organic unspecified (Extrinsic Allergic Alveolitis SOE; Hypersensitivity pneumonitis NOS (J67.0)

XXII - Bronchitis and pneumonitis due to chemicals, gases, fumes and vapors ("Acute Bronchitis Chemistry") (J68.0)

XXIII - Acute Pulmonary Edema due to chemicals, gases, fumes and vapors (Edema Pulmonary Chemical) (J68.1)

1. Beryllium and its toxic compounds (X49. -; Z57.5) (Table IV)

2. Bromine (X49. -; Z57.5) (Table V)

3. Cadmium or its compounds (X49. -; Z57.5) (Table VI)

4. Chlorine Gas (X47. -; Z57.5) (Table IX)

5. Fluorine or its toxic compounds (X47. -; Z57.5) (Table XI)

6. Halogen solvents respiratory irritants (X46. -; Z57.5) (Table XIII)

7. Iodine (X49. -; Z57.5) (Table XIV)

8. Manganese and its toxic compounds (X49. -; Z57.5) (Table XV)

9. Hydrogen cyanide (X47. -; Z57.5) (Table XVII)

1. Beryllium and its toxic compounds (X49. -; Z57.5) (Table IV)

2. Bromine (X49. -; Z57.5) (Table V)

3. Cadmium or its compounds (X49. -; Z57.5) (Table VI)

4. Chlorine Gas (X47. -; Z57.5) (Table IX)

5. Fluorine and its compounds (X47. -; Z57.5) (Table XI)

6. Halogen solvents respiratory irritants

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
XXIV - Syndrome Reactive Airway IX) Dysfunction (SDVA / RADS) (J68.3)	(X46. -; Z57.5) (Table XIII) 7. Iodine (X49. -; Z57.5) (Table XIV) 8. Hydrogen cyanide (X47. -; Z57.5) (Table XVII) 1. Bromine (X49. -; Z57.5) (Table V) 2. Cadmium or its compounds (X49. -; Z57.5) (Table VI) 3. Chlorine Gas (X47. -; Z57.5) (Table IX) 4. Halogenated Solvents respiratory irritants (X46. -; Z57.5) (Table XIII) 5. Iodine (X49. -; Z57.5) (Table XIV) 6. Hydrogen cyanide (X47. -; Z57.5) (Table XVII) 7. Ammonia (X49. -; Z57.5) 1. Arsenic and arsenic compounds (X49. -; Z57.4 and Z57.5) (Table I) 2. Beryllium and its compounds (X49. -; Z57.5) (Table IV) 3. Bromine (X49. -; Z57.5) (Table V) 4. Cadmium or its compounds (X49. -; Z57.5) (Table VI) 5. Chlorine Gas (X47. -; Z57.5) (Table IX)
XXV - Afecções due to chronic respiratory inhalation of gases, fumes, vapors and chemicals: Bronchiolitis Obliterans, Chronic, Chronic Diffuse Emphysema, Chronic Pulmonary Fibrosis (J68.4)	6. Fluorine and its compounds (X47. -; Z57.5) (Table XI) 7. Halogenated Solvents respiratory irritants (X46. -; Z57.5) (Table XIII) 8. Iodine (X49. -; Z57.5) (Table XIV) 9. Manganese and its toxic compounds (X49. -; Z57.5) (Table XV) 10. Hydrogen cyanide (X47. -; Z57.5) (Table XVII) 11. Hydrogen sulfide (hydrogen sulfide) (X47. -; Z57.5) (Table XVII) 12. Carbides of heavy metals (X49. -; Z57.5) 13. Ammonia (X49. -; Z57.5) 14. Sulfur dioxide (X49. -; Z57.5)

DISEASES

Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE

15. Mists and aerosols of mineral acids (X47. -; Z57.5)

16. Acrylates (X49. -; Z57.5)

17. Selenium and compounds (X49. -; Z57.5)

XXVI - Radiation Pneumonitis

(acute event) (J70.0) and Pulmonary Fibrosis Consequent Radiation (chronic manifestation) (J70.1)

Ionizing radiation (W88. -; Z57.1) (Table XXIV)

XXVII - Pleural effusion (J90. -)

Occupational exposure to dust from asbestos or asbestos (Z57.2) (Table II)

XXVIII - Pleural plaques (J92. -)

Occupational exposure to dust from asbestos or asbestos (Z57.2) (Table II)

XXIX - interstitial emphysema (J98.2)

Cadmium or its compounds (X49. -; Z57.5) (Table VI)

XXX - Respiratory disorders in other systemic connective tissue diseases classified elsewhere (M05.3), "Caplan's syndrome (J99.1)

1. Occupational exposure to dust Coal (Z57.2)

2. Occupational exposure to silica dust free (Z57.2) (Table XVIII)

DISEASES OF THE DIGESTIVE SYSTEM RELATED WORK (Group XI of ICD-10)

DISEASES

Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE

I - Tooth Erosion (K03.2)

1. Mists of fluoride or toxic compounds (X47. -; Z57.5) (Table XI)

2. Occupational exposure to other mists acidic (X47. -; Z57.5)

II - post-eruptive changes the color of the hard tissues of teeth (K03.7)

1. Mists of Cadmium and its compounds (X47. -; Z57.5) (Table VI)

2. Occupational exposure to metals: Copper, Nickel, Silver (X47. -; Z57.5)

III - Chronic Gingivitis (K05.1)

Mercury and its toxic compounds (X49. -; Z57.5) (Table XVI)

IV - Chronic ulcerative stomatitis (K12.1)

1. Arsenic and arsenic compounds (X49. -; Z57.5) (Table I)

2. Bromine (X49. -; Z57.5) (Table XII)

3. Mercury and its toxic compounds (X49. -; Z57.5) (Table XVI)

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
V - Gastroenteritis and Colitis toxic (K52. -)	1. Arsenic and arsenic compounds (X49. -; Z57.5) (Table I) 2. Cadmium or its compounds (X49. -; Z57.5) (Table VI) 3. Ionizing radiation (W88. -; Z57.1) (Table XXIV)
VI - Other functional intestinal disorders (abdominal pain syndrome paroxysmal afebrile, with state subocclusive ("lead colic") (K59.8)	Lead or its toxic compounds (X49. -; Z57.5) (Table VIII)
VII - Toxic Liver Disease (K71. -): Toxic Liver Disease, Liver with necrosis (K71.1) Toxic Liver Disease with Acute hepatitis (K71.2) Toxic Liver disease with chronic persistent hepatitis (K71.3); Toxic Liver Disease with Liver Disorders (K71.8)	1. Vinyl chloride, chlorobenzene, carbon tetrachloride, chloroform and other halogenated solvents hepatotoxic (X46. - and X48. -, Z57.4 and Z57.5) (Table XIII) 2. Hexachlorobenzene (HCB) (X48. -; Z57.4 and Z57.5) 3. Polychlorinated biphenyls (PCBs) (X49. -; Z57.4 and Z57.5) 4. Tetrachlorodibenzodioxin (TCDD) (X49. -) 1. Arsenic and arsenic compounds (X49. -; Z57.4 and Z57.5) (Table I) 2. Vinyl Chloride (X46. -; Z57.5) (Table XIII) 3. Thorium (X49. -; Z57.5)
VIII - Portal Hypertension (K76.6)	

**DISEASES OF THE SKIN AND SUBCUTANEOUS TISSUE
RELATED WORK (Group XII of ICD-10)**

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
I - Other Local Infections Skin and Subcutaneous Tissue, "Dermatoses popular and pustulous and its infectious complications (L08.9)	1. Chromium and its toxic compounds (Z57.5) (Table X) 2. Aliphatic or aromatic hydrocarbons (toxic derivatives) (Z57.5) (Table XIII) 3. Microorganisms live and infectious parasites and their toxic products (Z57.5) (Table XXV) 4. Other chemical or biological agents that affect the skin, not included under

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
	other headings (Z57.5) (Table XXVII)
II - Allergic Contact Dermatitis due to metals (L23.0)	1. Chromium and its toxic compounds (Z57.5) (Table X)
III - Allergic Contact Dermatitis due to Adhesives (L23.1)	2. Mercury and its toxic compounds (Z57.5) (Table XVI)
IV - Allergic Contact Dermatitis due to cosmetics (manufacture / handling) (L23.2)	Adhesives in occupational exposure (Z57.5) (Table XXVII)
V - Allergic Contact Dermatitis due to drugs in contact with the skin (L23.3)	Manufacturing / handling Cosmetics (Z57.5) (Table XXVII)
VI - Allergic Contact Dermatitis due to dyes (L23.4)	Drugs, occupational exposure (Z57.5) (Table XXVII)
VII - Allergic Contact Dermatitis due to other products chemicals (L23.5)	Dyes, for occupational exposure (Z57.5) (Table XXVII)
	1. Chromium and its toxic compounds (Z57.5) (Table X)
	2. Phosphorus or their toxic products (Z57.5) (Table XII)
	3. Iodine (Z57.5) (Table XIV)
	4. Tar, Pitch, Bitumen, Coal Mining, paraffin or residues of these substances (Z57.8) (Table XX)
	5. Rubber (Z57.8) (Table XXVII)
	6. Insecticides (Z57.5) (Table XXVII)
	7. Plastics (Z57.8) (Table XXVII)
VIII - Allergic Contact Dermatitis due to food in contact with the skin (manufacturing / handling) (L23.6)	Manufacturing / Food handling (Z57.5) (Table XXVII)
IX - Allergic Contact Dermatitis due to plants (not including plants used as food) (L23.7)	Handling Plants for occupational exposure (Z57.8) (Table XXVII)
X - Allergic Contact Dermatitis due to other agents (External Cause specified) (L23.8)	Chemicals NES in occupational exposure (Z57.5) (Table XXVII)
XI - Irritant contact dermatitis due to detergents (L24.0)	Detergents in occupational exposure (Z57.5) (Table XXVII)
XII - Irritant contact dermatitis	Oils and Fats in occupational exposure

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
due to oils and fats (L24.1)	(Z57.5) (Table XXVII)
XIII - Irritant contact dermatitis due to solvents: ketones, Cyclohexane, Chlorine Compounds, Esters, Glycol, Hydrocarbons (L24.2)	1. Benzene (X46. -; Z57.5) (Table III) 2. Aliphatic or aromatic hydrocarbon or halogenated toxic (Z57.5) (Table XIII)
XIV - Irritant contact dermatitis due to cosmetics (L24.3)	Cosmetics in occupational exposure (Z57.5) (Table XXVII)
XV - Contact Dermatitis by Irritants due to drugs in contact with the skin (L24.4)	Drugs, occupational exposure (Z57.5) (Table XXVII)
XVI - Irritant contact dermatitis due to other chemicals: Arsenic, Beryllium, Bromine, Chromium, Cement, Fluoride, Phosphorus, Insecticides (L24.5)	1. Arsenic and arsenic compounds (Z57.5) (Table I) 2. Beryllium and its toxic compounds (Z57.5) (Table IV) 3. Bromine (Z57.5) (Table V) 4. Chromium and its toxic compounds (Z57.5) (Table X) 5. Fluorine or its toxic compounds (Z57.5) (Table XI) 6. Phosphorus (Z57.5) (Table XII)
XVII - Irritant contact dermatitis due to food in contact with the skin (L24.6)	Foods, occupational exposure (Z57.8) (Table XXVII)
XVIII - Irritant contact dermatitis due to plants except food (L24.7)	Plants for occupational exposure (Z57.8) (Table XXVII)
XIX - Irritant contact dermatitis due to other agents: Dyes (L24.8)	Chemicals NES in occupational exposure (Z57.5) (Table XXVII)
XX - Allergic urticaria (L50.0)	Pesticides and other chemicals (X48. -; Z57.4 and Z57.5) (Table XXVII)
XXI - Urticaria due to Heat and Cold (L50.2)	Occupational exposure to heat and cold (W92, -; W93 .-, Z57.6) (Table XXVII) Occupational exposure to chemical, physical and biological processes that affect the skin (X49. -; Z57.4 and Z57.5)
XXII - Contact urticaria (L50.6)	

DISEASES

**Etiologic agent or RISK FACTORS
OF OCCUPATIONAL NATURE**

(Table XXVII)

XXIII - Sunburn (L55)

Occupational exposure to actinic radiation (X32. -; Z57.1) (Table XXVII)

XXIV - Other acute skin changes due to Ultraviolet Radiation

(L56. -): Dermatitis by

photocontact (Berloque

Dermatitis) (L56.2) Urticaria

Solar (L56.3) Other specified

acute changes of the skin due to

Ultraviolet Radiation (L56.8)

Other acute skin changes due to

Ultraviolet Radiation, Not

Otherwise Specified (L56 .9);

Ultraviolet Radiation (W89. -; Z57.1) (Table XXVII)

XXV - Skin changes due to

Chronic Exposure to Ionizing

Radiation (L57. -): Actinic

Keratosis (L57.0) Other

Changes: Solar dermatitis,

"Farmer's Skin," "Skin Mariner"

(L57.8)

XXVI - Radiodermatitis (L58. -):

Acute Radiodermatitis (L58.0);

Radiodermatitis Chronicle

(L58.1); Radiodermatitis,

unspecified (L58.9), Skin and

connective tissue related to

radiation, unspecified (L59.9)

Non-ionizing radiation (W89. -; X32 .-, Z57.1) (Table XXVII)

Ionizing radiation (W88. -; Z57.1) (Table XXIV)

XXVII - Other forms of Acne:

"Chloracne" (L70.8)

1. Halogenated aromatic hydrocarbons, mono, Monobromobenzene, Hexachlorobenzene (X46.; Z57.5) (Table XIII)

2. Derivatives of phenol, pentachlorophenol and hidrobenzonitrilo (X49. -; Z57.4 and Z57.5) (Table XXVII)

3. Polychlorinated biphenyls (PCBs)

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE (X49. -; Z57.4 and Z57.5) (Table XXVII)
XXVIII - Other forms of Follicular Cysts Skin and Subcutaneous tissue, "Elaiioconiose" or "Follicular dermatitis (L72.8)	Oils and fats or synthetic mineral (X49. -; Z57.5) (Table XXVII)
XXIX - Other forms of hyperpigmentation by melanin: "melanosis" (L81.4)	<ol style="list-style-type: none"> 1. Arsenic and arsenic compounds (X49. -; Z57.4 and Z57.5) (Table I) 2. Chlorobenzene and dichlorobenzene (X46. -; Z57.4 and Z57.5) (Table XIII) 3. Tar, Pitch, Bitumen, Coal Mining, paraffin, creosote, tar, Coal tar residues or substances (Z57.8) (Table XX) 4. Anthracene and Dibenzoanthracene (Z57.5) (Table XX) 5. Bismuth (X44. -; Z57.5) (Table XXVII) 6. Cytostatic (X44. -; Z57.5) (Table XXVII) 7. Nitrogen compounds: Nitric acid, Dinitrophenol (X49. -; Z57.5) (Table XXVII) 8. Naphthols added to dyes (X49. -; Z57.5) (Table XXVII) 9. Oils Cutting (Z57.5) (Table XXVII) 10. Parafenilenodiamina and its derivatives (X49. -; Z57.5) (Table XXVII) 11. Dust of certain woods (Z57.3) (Table XXVII) 12. Quinine and its derivatives (Z57.5) (Table XXVII) 13. Gold salts (X44. -; Z57.5) (Table XXVII) 14. Silver salts (Sequelae of Chronic Contact Dermatitis) (X44. -; Z57.5) (Table XXVII)
XXX - leukoderma, not	1. Arsenic and compounds (X49. -;

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
elsewhere classified (Includes "Occupational Vitiligo") (L81.5)	Z57.4 and Z57.5) (Table I) 2. Hydroquinone derivatives and esters (X49. -; Z57.5) (Table XXVII) 3. Monomethyl ether of hydroquinone (Mbeh) (X49. -; Z57.5) (Table XXVII) 4. To-Aminophenol (X49. -; Z57.5) (Table XXVII) 5. To-butylphenol (X49. -; Z57.5) (Table XXVII) 6. Para-Cresol (X49. -; Z57.5) (Table XXVII) 7. Catechol and pyrocatechol (X49. -; Z57.5) (Table XXVII) 8. Chlorophenol (X46. -; Z57.4 and Z57.5) (Table XXVII) Halogenated aromatic hydrocarbons: minocloro-benzene, benzene-monobromo, hexachlorobenzene (X46. -; Z57.4 and Z57.5) (Table XIII)
XXXI - Other specified disorders of pigmentation: "Porphyria Cutaneous Late" (L81.8)	Arsenic and arsenic compounds (X49. -; Z57.4 and Z57.5) (Table I)
XXXII - Acquired Palmoplantar Keratosis (L85.1)	1. Chromium and its toxic compounds (Z57.5) (Table X) 2. Enzymes of animal, plant or bacterial (Z57.8) (Table XXVII)
XXXIII - Chronic Ulcer of Skin, not elsewhere classified (L98.4)	1. Ethyl Chloride (local anesthetic) (W93. -; Z57.6) (Table XIII) 2. Frio (X31. -; W93. -, Z57.6) (Table XXVII)
XXXIV - Frostbite (Frostbite) Superficial (T33): Erythema pernio	1. Ethyl Chloride (local anesthetic) (W93. -; Z57.6) (Table XIII) 2. Frio (X31. -; W93. -, Z57.6) (Table XXVII)
XXXV - Frostbite (Frostbite) with Tissue Necrosis (T34)	
Musculoskeletal diseases	TISSUE AND, IN CONNECTION WITH THE WORK

(Group XIII of ICD-10)

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
I - Rheumatoid arthritis associated	1. Occupational exposure to coal dust

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
with Coal Workers' Pneumoconiosis (Z57.2) (J60. -), "Caplan's syndrome" (M05.3)	2. Occupational exposure to silica dust (Z57.2) (Table XVIII)
II - Gout induced by lead (M10.1)	Lead or its toxic compounds (X49. -; Z57.5) (Table VIII)
III - Other Arthrosis (M19. -)	Awkward positions and repetitive movements (Z57.8)
IV - Other joint disorders not elsewhere classified: Articular Pain (M25.5)	1. Positions forced and repetitive gestures (Z57.8) 2. Vibrations localized (W43. -; Z57.7) (Table XXII)
V - cervicobrachial syndrome (M53.1)	1. Positions forced and repetitive gestures (Z57.8) 2. Vibrations localized (W43. -; Z57.7) (Table XXII)
VI - Back pain (M54. -): Neck (M54.2) Sciatica (M54.3) Lumbago with sciatica (M54.4)	1. Positions forced and repetitive gestures (Z57.8) 2. Pattern of drudgery (Z56.3) 3. Difficult working conditions (Z56.5)
VII - Synovitis and tenosynovitis (M65. -) Trigger Finger (M65.3), the radial styloid tenosynovitis (De Quervain) (M65.4) Other synovitis and tenosynovitis (M65.8), synovitis and tenosynovitis, unspecified (M65.9)	1. Positions forced and repetitive gestures (Z57.8) 2. Pattern of drudgery (Z56.3) 3. Difficult working conditions (Z56.5)
VIII - Soft tissue disorders related to the use, overuse and pressure, resulting from occupational (M70. -): Sizzling Synovitis Chronic hand and wrist (M70.0) Bursitis of the Hand (M70.1) Bursitis olecranon (M70.2) Other bursitis of the elbow (M70.3) Other Pre-patellar bursitis (M70.4) Other bursitis of the knee (M70.5) Other Soft tissue disorders related to the use, overuse and pressure (M70.8) Disorder Unspecified soft tissue related to the	1. Positions forced and repetitive gestures (Z57.8) 2. Pattern of drudgery (Z56.3) 3. Difficult working conditions (Z56.5)

DISEASES

Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE

use, overuse and pressure (M70.9).

IX - Fibromatosis of the Palmar
Fascia: "Dupuytren's Contracture or
Illness" (M72.0)

1. Positions forced and repetitive gestures (Z57.8)
2. Vibrations localized (W43. -; Z57.7) (Table XXII)

X - Shoulder Injury (M75. -):
Adhesive capsulitis of the shoulder
(frozen shoulder, periarthritis of the
shoulder) (M75.0) syndrome or
rotator cuff supraspinatus syndrome
(M75.1), tendinitis (M75.2) ;
Calcifying tendinitis of shoulder
(M75.3) Shoulder bursitis (M75.5)
Other Shoulder Injuries (M75.8),
Shoulder Injury, unspecified
(M75.9)

1. Positions forced and repetitive gestures (Z57.8)
2. Pattern of drudgery (Z56)
3. Vibrations localized (W43. -; Z57.7) (Table XXII)

XI - Other enthesopathies (M77. -)
Medial epicondylitis (M77.0)
Lateral epicondylitis (Tennis
Elbow), myalgia (M79.1)

1. Positions forced and repetitive gestures (Z57.8)
2. Vibrations localized (W43. -; Z57.7) (Table XXII)

XII - Other specified soft tissue
disorders (M79.8)

1. Positions forced and repetitive gestures (Z57.8)
2. Vibrations localized (W43. -; Z57.7) (Table XXII)

XIII - Adult osteomalacia induced
by drugs (M83.5)

1. Cadmium or its compounds (X49. -) (Table VI)
2. Phosphorus and its compounds (Sesquissulfeto Phosphorus) (X49. -; Z57.5) (Table XII)

XIV - Skeletal Fluorosis (M85.1)

- Fluorine and its toxic compounds (X49. - ; Z57.5) (Table XI)

XV - Osteonecrosis (M87. -):
Osteonecrosis due to drugs (M87.1)
Other secondary osteonecrosis
(M87.3)

1. Phosphorus and its compounds (Sesquissulfeto Phosphorus) (X49. -; Z57.5) (Table XII)
2. Vibrations localized (W43. -; Z57.7) (Table XXII)
3. Ionizing radiation (Z57.1) (Table

DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
	XXIV)
XVI - Osteolysis (M89.5) (of the distal phalanges of the fingers)	Vinyl Chloride (X49. -; Z57.5) (Table XIII)
XVII - Osteonecrosis in "Evil of Coffins" (M90.3)	"Air Power" (W94. -; Z57.8) (Table XXIII)
XVIII - Kienböck Disease for Adults (Adult Osteo-condrose the Carpal Lunate) (M93.1) and other osteochondral patias-unspecified (M93.8)	Localized vibrations (W43. -; Z57.7) (Table XXII)
DISEASES OF THE genito-urinary system RELATED WORK (Group XIV of the ICD-10)	
DISEASES	Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE
I - acute nephritic syndrome (N00. -)	Nephrotoxic halogenated aliphatic hydrocarbons (X46. -; Z57.5) (Table XIII)
II - Chronic Glomerular Disease (N03. -)	Mercury and its toxic compounds (X49. -; Z57.5) (Table XVI)
III - tubulointerstitial nephropathy induced by heavy metals (N14.3)	1. Cadmium or its compounds (X49. -; Z57.5) (Table VI) 2. Lead or its toxic compounds (X49. -; Z57.5) (Table VIII) 3. Mercury and its toxic compounds (X49. -; Z57.4 and Z57.5) (Table XVI) Nephrotoxic halogenated aliphatic hydrocarbons (X46. -; Z57.5) (Table XIII)
IV - Acute Renal Failure (N17)	Lead and its compounds (X49. -; Z57.5) (Table VIII)
V - Chronic Renal Failure (N18)	Aromatic amines and their derivatives (X49. -; Z57.5)
VI - Acute cystitis (N30.0)	1. Lead or its toxic compounds (X49. -; Z57.5) (Table VIII) 2. Ionizing radiation (W88. -; Z57.1) (Table XXIV) 3. Chlordecone (X48. -; Z57.4) 4. Dibromochloropropane (DBCP) (X48. -; Z57.4 and Z57.5) 5. Heat (work in high temperatures) (Z57.6)
VII - Male Infertility (N46)	

**INJURIES, POISONING AND CERTAIN OTHER
CONSEQUENCES OF EXTERNAL CAUSES RELATED TO
WORK**

(Group XIX of ICD-10)

DISEASES

**Etiologic agent or RISK FACTORS
OF OCCUPATIONAL NATURE**

I - Toxic Effects of Organic Solvents
(T52. -): alcohols (T51.8) and
ketones (T52.4), Benzene, Toluene
and Xylene (T52.1 and T52.2)
Halogenated derivatives of aliphatic
and aromatic hydrocarbons (T53):
Carbon tetrachloride (T53.0),
Chloroform (T53.1),
Trichloroethylene (T53.2),
Tetrachloroethylene (T53.3),
dichloroethane (T53.4),
chlorofluorocarbons, carbon (T53.5)
Other halogenated aliphatic
hydrocarbons (T53.6) Other
halogenated aromatic hydrocarbons
(T53.7) Halogenated aliphatic
hydrocarbons and aromatic
hydrocarbons, unspecified (T53.9);
Carbon Sulfide (T65.4)

Occupational exposure to toxic agents in
other industries (Z57.5)

II - Toxic effect of corrosive
substances (T54): Phenol and
phenol homologues (T54.0),
Fluorine and its compounds
(T65.8) Selenium and its
compounds (T56.8) Other
corrosive organic compounds
(T54.1), acids and corrosive
substances like acid (T54.2),
caustic alkalis and alkali similar
(T54.3) Toxic effect of corrosive
substance, unspecified (T54.9).

Occupational exposure to toxic agents in
other industries (Z57.5)

III - Effect of toxic metals (T56):
Arsenic and compounds (T57.0)

Occupational exposure to toxic agents in
other industries (Z57.5)

DISEASES

Etiologic agent or RISK FACTORS OF OCCUPATIONAL NATURE

Cadmium and its compounds (T56.3) Lead and its compounds (T56.0), Chromium and compounds (T56.2); Manganese and its compounds (T57.2) Mercury and its compounds (T56.1) Other metals (T56.8); Metal, unspecified (T56.9).
IV - Asphyxiating Chemicals (T57-59): Carbon monoxide (T58), hydrocyanic acid and cyanides (T57.3), hydrogen sulfide (T59.6), aromatic amines and their derivatives (T65.3)
V - Pesticides (Pesticides, "Pesticide") (T60): Organophosphates and Carbamates (T60.0); Halogenated (T60.1) Other pesticides (T60.2)
VI - Effect of Air Pressure and Water Pressure (T70): Barotrauma Otitis (T70.0); Sinus Barotrauma (T70.1), decompression sickness ("Coffins of Evil") (T70.3) Other effects of air pressure and water (T70.8).

Occupational exposure to toxic agents in other industries (Z57.5)

Occupational exposure to toxic agents in agriculture (Z57.4)

Occupational exposure to abnormal atmospheric pressures (W94. -; Z57.8)

LIST C

Note:

1 - intervals are indicated ICD-10 which recognizes Nexus Technical Epidemiological, in accordance with § 3 of art. 337, between the disease entity and the class of NCEA indicated, all subclasses included therein whose four digits are common.

INTERVAL ICD-10

NCEA

A15-A19	0810	1091	1411	1412	1533	1540	2330	3011	3701
	3702	3811	3812	3821	3822	3839	3900	4120	4211
	4213	4222	4223	4291	4299	4312	4321	4391	4399
	4687	4711	4713	4721	4741	4742	4743	4744	4789
	4921	4923	4924	4929	5611	7810	7820	7830	8121
	8122	8129	8610	9420	9601				

INTERVAL ICD-10

NCEA

	1091 3600 3701 3702 3811 3812 3821 3822 3839
	3900 4120 4211 4213 4222 4223 4291 4292 4299
E10-E14	4313 4319 4329 4399 4721 4921 4922 4923 4924
	4929 4930 5030 5231 5239 8011 8012 8020 8030
	8121 8122 8129 8411 9420

INTERVAL ICD-10

NCEA

	0710 0990 1011 1012 1013 1220 1532 1622 1732
	1733 2211 2330 2342 2451 2511 2512 2531 2539
	2542 2543 2593 2814 2822 2840 2861 2866 2869
	2920 2930 3101 3102 3329 3600 3701 3702 3811
F10-F19	3812 3821 3822 3839 3900 4120 4211 4213 4221
	4292 4299 4313 4319 4321 4329 4399 4520 4912
	4921 5030 5212 5221 5222 5223 5229 5231 5232
	5239 5250 5310 6423 7810 7820 7830 8121 8122
	8129 8411 8423 8424 9420

	0710 0990 1011 1012 1013 1031 1071 1321 1411
	1412 2330 2342 2511 2543 2592 2861 2866 2869
	2942 3701 3702 3811 3812 3821 3822 3839 3900
F20-F29	4120 4211 4213 4222 4223 4291 4292 4299 4312
	4391 4399 4921 4922 4923 4924 4929 5212 5310
	6423 7732 7810 7820 7830 8011 8012 8020 8030
	8121 8122 8129 8423 9420

	0710 0892 0990 1011 1012 1013 1031 1220 1311
	1313 1314 1321 1330 1340 1351 1359 1411 1412
	1413 1422 1531 1532 1540 2091 2123 2511 2710
	2751 2861 2930 2945 3299 3600 4636 4711 4753
F30-F39	4756 4759 4762 4911 4912 4921 4922 4923 4924
	4929 5111 5120 5221 5222 5223 5229 5310 5620
	6110 6120 6130 6141 6142 6143 6190 6311 6422
	6423 6431 6550 8121 8122 8129 8411 8413 8423
	8424 8610 8711 8720 8730 8800

	0710 0990 1311 1321 1351 1411 1412 1421 1532
	2945 3600 4711 4753 4756 4759 4762 4911 4912
	4921 4922 4923 4924 4929 5111 5120 5221 5222
F40-F48	5223 5229 5310 6110 6120 6130 6141 6142 6143
	6190 6311 6422 6423 8011 8012 8020 8030 8121
	8122 8129 8411 8423 8424 8610

INTERVAL ICD-10

NCEA

G40-G47	0113 0210 0220 0810 1011 1012 1013 1321 1411
	1412 1610 1621 1732 1733 1931 2330 2342 2511

INTERVAL ICD-10

NCEA

A15-A19	0810 1091 1411 1412 1533 1540 2330 3011 3701
	3702 3811 3812 3821 3822 3839 3900 4120 4211
	4213 4222 4223 4291 4299 4312 4321 4391 4399
	4687 4711 4713 4721 4741 4742 4743 4744 4789
	4921 4923 4924 4929 5611 7810 7820 7830 8121
	8122 8129 8610 9420 9601

INTERVAL ICD-10

NCEA

G50-G59	2539 2861 3701 3702 3811 3812 3821 3822 3839
	3900 4120 4211 4213 4222 4223 4291 4292 4299
	4313 4319 4399 4921 4922 4923 4924 4929 4930
	5212 8011 8012 8020 8030 8121 8122 8129
	0155 1011 1012 1013 1062 1093 1095 1313 1351
	1411 1412 1421 1529 1531 1532 1533 1539 1540
	2063 2123 2211 2222 2223 2229 2349 2542 2593
	2640 2710 2759 2944 2945 3240 3250 4711 5611
	5612 5620 6110 6120 6130 6141 6142 6143 6190
	6422 6423 8121 8122 8129 8610

INTERVAL ICD-10

NCEA

H53-H54	0210 0220 0810 1071 1220 1610 1622 2330 2342
	3701 3702 3811 3812 3821 3822 3839 3900 4120
	4211 4212 4213 4222 4223 4291 4299 4312 4313
	4319 4321 4329 4391 4399 4741 4742 4743 4744
	4789 4921 4922 4923 4924 4929 4930 8011 8012
	8020 8030 8121 8122 8129

INTERVAL ICD-10

NCEA

I05-I09	4921
I10-I15	0111 1411 1412 4921 4922 4923 4924 4929 5111
	5120
I20-I25	1621 4120 4211 4213 4221 4222 4223 4291 4299
	4329 4399 4921 4922 4930 6110 6120 6130 6141
I30-I52	6142 6143 6190
	0113 0210 0220 0810 1011 1012 1013 1061 1071
	1411 1412 1610 1931 2029 2330 2342 3600 3701
	3702 3811 3812 3821 3822 3839 3900 4120 4211
	4213 4222 4223 4291 4292 4299 4312 4313 4319
	4391 4399 4621 4622 4623 4921 4922 4923 4924
	4929 4930 8121 8122 8129 8411 9420
I60-I69	0810 1071 2330 2342 3600 3701 3702 3811 3812
	3821 3822 3839 3900 4120 4211 4213 4222 4223
	4291 4299 4312 4313 4319 4321 4391 4399 4921

INTERVAL ICD-10

NCEA

A15-A19
 0810 1091 1411 1412 1533 1540 2330 3011 3701
 3702 3811 3812 3821 3822 3839 3900 4120 4211
 4213 4222 4223 4291 4299 4312 4321 4391 4399
 4687 4711 4713 4721 4741 4742 4743 4744 4789
 4921 4923 4924 4929 5611 7810 7820 7830 8121
 8122 8129 8610 9420 9601

INTERVAL ICD-10

NCEA

I80-I89
 4922 4923 4924 4929 4930 8112 8121 8122 8129
 8411 8591 9200 9311 9312 9313 9319 9420
 1011 1012 1013 1020 1031 1033 1091 1092 1220
 1311 1321 1351 1411 1412 1413 1422 1510 1531
 1532 1540 1621 1622 2123 2342 2542 2710 2813
 2832 2833 2920 2930 2944 2945 3101 3102 3329
 3701 3702 3811 3812 3821 3822 3839 3900 4621
 4622 4623 4721 4722 4921 4922 5611 5612 5620
 8011 8012 8020 8030 8121 8122 8129 8411 8610
 9420 9491 9601

INTERVAL ICD-10

NCEA

J40-J47
 0810 1031 1220 1311 1321 1351 1411 1412 1610
 1622 1629 2330 2342 2539 3101 3102 3329 4120
 4211 4213 4292 4299 4313 4319 4399 4921 8121
 8122 8129 8411

INTERVAL ICD-10

NCEA

K35-K38
 0810 1011 1012 1013 1071 1411 1412 1531 1540
 1610 1621 1732 1733 2451 2511 2512 2832 2833
 2930 3101 3329 4621 4622 4623 4921 4922 8610
 0113 0210 0220 0230 0810 1011 1012 1013 1020
 1031 1033 1041 1051 1061 1066 1071 1091 1122
 1321 1354 1510 1610 1621 1622 1629 1722 1732
 1733 1931 2211 2212 2219 2330 2341 2342 2349
 2443 2449 2451 2511 2512 2521 2539 2541 2542
 2543 2592 2593 2710 2815 2822 2832 2833 2861
 K40-K46
 2866 2869 2930 2943 2944 2945 3011 3101 3102
 3329 3701 3702 3811 3812 3821 3822 3839 3900
 4120 4211 4212 4213 4221 4222 4223 4291 4292
 4299 4312 4313 4319 4321 4329 4391 4399 4621
 4622 4623 4632 4634 4687 4721 4722 4741 4742
 4743 4744 4789 4921 4922 4930 5212 8121 8122
 8129 9420

INTERVAL ICD-10

NCEA

Tratado de Toxicologia Ocupacional

INTERVAL ICD-10

NCEA

A15-A19	0810	1091	1411	1412	1533	1540	2330	3011	3701
	3702	3811	3812	3821	3822	3839	3900	4120	4211
	4213	4222	4223	4291	4299	4312	4321	4391	4399
	4687	4711	4713	4721	4741	4742	4743	4744	4789
	4921	4923	4924	4929	5611	7810	7820	7830	8121
	8122	8129	8610	9420	9601				

INTERVAL ICD-10

NCEA

L60-L75	8610								
L80-L99	0113	1011	1012	1013	1071	1411	1412	1610	1621
	1931	2451	5611	5620	8121	8122	8129	8610	

INTERVAL ICD-10

NCEA

M00-M25	0113	0131	0133	0210	0220	0810	0892	0910	1011
	1012	1013	1020	1031	1033	1041	1051	1052	1061
	1064	1071	1072	1091	1122	1220	1311	1321	1351
	1354	1411	1412	1413	1532	1621	1732	1733	1931
	2012	2019	2312	2330	2341	2342	2349	2431	2443
	2449	2511	2522	2539	2543	2550	2710	2813	2815
	2822	2852	2853	2854	2861	2862	2865	2866	2869
	2920	2930	2944	2945	2950	3011	3102	3600	3701
	3702	3811	3812	3821	3822	3839	3900	4120	4211
	4212	4213	4221	4222	4223	4291	4292	4299	4312
	4313	4319	4321	4329	4391	4399	4621	4622	4623
	4636	4661	4711	4721	4921	4922	4923	4924	4929
	4930	5012	5021	5212	5310	5611	5620	7719	8121
	8122	8129	8411	8424	8430	8591	8610	9200	9311
	9312	9313	9319	9420	9491	9601			
M30-M36	1412	8121	8122	8129	8610				
	0113	0131	0133	0210	0220	0230	0500	0710	0810
	0892	0910	0990	1011	1012	1013	1020	1031	1033
	1041	1051	1052	1061	1062	1064	1071	1072	1092
M40-M54	1122	1311	1312	1321	1323	1340	1351	1354	1411
	1412	1413	1421	1422	1510	1532	1610	1621	1622
	1623	1629	1710	1721	1722	1732	1733	1931	2012
	2019	2029	2040	2091	2093	2123	2211	2212	2219
	2221	2222	2312	2320	2330	2341	2342	2349	2391
	2431	2439	2441	2443	2449	2451	2511	2513	2521
	2522	2539	2542	2543	2550	2592	2593	2710	2722
	2733	2813	2815	2822	2832	2833	2852	2853	2854
	2861	2862	2864	2866	2869	2920	2930	2942	2943
	2944	2945	2950	3011	3101	3102	3240	3321	3329

INTERVAL ICD-10

NCEA

A15-A19	0810	1091	1411	1412	1533	1540	2330	3011	3701
	3702	3811	3812	3821	3822	3839	3900	4120	4211
	4213	4222	4223	4291	4299	4312	4321	4391	4399
	4687	4711	4713	4721	4741	4742	4743	4744	4789
	4921	4923	4924	4929	5611	7810	7820	7830	8121
	8122	8129	8610	9420	9601				

INTERVAL ICD-10

NCEA

M60-M79	3600	3701	3702	3811	3812	3821	3822	3839	3900
	4120	4211	4212	4213	4222	4223	4291	4292	4299
	4311	4312	4313	4319	4321	4329	4391	4399	4621
	4622	4623	4632	4636	4661	4681	4682	4685	4686
	4687	4689	4921	4922	4923	4924	4929	4930	5012
	5021	5211	5212	5221	5222	5223	5229	5310	5612
	5620	6431	7719	7732	8121	8122	8129	8424	8430
	8610	9420							
	0113	0155	0210	0220	1011	1012	1013	1020	1031
	1033	1051	1052	1062	1064	1092	1093	1094	1095
	1096	1099	1122	1311	1314	1321	1323	1340	1351
	1352	1354	1359	1411	1412	1413	1414	1421	1510
	1521	1529	1531	1532	1533	1540	1623	1732	1733
	1742	1749	2040	2063	2091	2110	2121	2123	2211
	2219	2221	2222	2223	2229	2312	2319	2342	2349
	2439	2443	2449	2451	2531	2539	2541	2542	2543
	2550	2591	2592	2593	2610	2631	2632	2640	2651
	2710	2721	2722	2732	2733	2740	2751	2759	2813
	2814	2815	2822	2823	2824	2840	2853	2854	2861
	2864	2866	2869	2920	2930	2941	2942	2943	2944
	2945	2949	3092	3101	3102	3104	3230	3240	3250
	3291	3299	3316	3329	3701	3702	3811	3812	3821
	3822	3839	3900	4221	4632	4634	4711	4713	4912
	5111	5120	5212	5221	5222	5223	5229	5310	5320
	5612	5620	6021	6022	6110	6120	6130	6141	6142
	6143	6190	6209	6311	6399	6422	6423	6431	6550
	7410	7490	7719	7733	8121	8122	8129	8211	8219
	8220	8230	8291	8292	8299	8610	9420	9601	

INTERVAL ICD-10

NCEA

S00-S09	0210	0220	0230	0810	1011	1012	1013	1033	1041
	1061	1071	1122	1321	1510	1532	1610	1621	1622
	1732	1733	1931	2212	2330	2342	2391	2511	2512

Tratado de Toxicología Ocupacional

INTERVAL ICD-10

NCEA

	2539	2542	2543	2593	2832	2833	2866	2869	2930
	3011	3101	3102	3329	3701	3702	3811	3812	3821
	3822	3839	3900	4120	4211	4213	4221	4222	4223
	4291	4292	4299	4312	4313	4319	4321	4329	4391
	4399	4520	4530	4541	4542	4621	4622	4623	4635
	4671	4672	4673	4674	4679	4687	4731	4732	4741
	4742	4743	4744	4789	4921	4922	4930	5212	5320
	7810	7820	7830	8011	8012	8020	8030	8121	8122
	8129	9420							
	0113	0131	0133	0210	0220	0230	0810	1011	1012
	1013	1071	1321	1510	1610	1621	1622	1629	1732
	1733	1931	2330	2342	2512	2539	2543	2832	2833
S20-S29	2866	2869	3600	3701	3702	3811	3812	3821	3822
	3839	3900	4120	4211	4213	4221	4222	4223	4291
	4292	4299	4321	4399	4621	4622	4623	4632	4687
	4741	4742	4743	4744	4789	4921	4922	4930	5212
	5310	8121	8122	8129	9420				
	0131	0133	0210	0220	1011	1012	1013	1061	1071
	1610	1621	2330	2342	2511	2512	3101	3329	3701
	3702	3811	3812	3821	3822	3839	3900	4120	4211
S30-S39	4213	4221	4222	4223	4291	4299	4312	4313	4319
	4321	4329	4391	4399	4621	4622	4623	4687	4722
	4741	4742	4743	4744	4789	4921	4930	5212	5221
	5222	5223	5229	7810	7820	7830	8121	8122	8129
	9420								
	0131	0133	0210	0220	0500	0810	1011	1012	1013
	1031	1033	1041	1051	1061	1064	1071	1091	1122
	1321	1351	1354	1411	1412	1510	1531	1532	1533
	1540	1610	1621	1622	1623	1629	1722	1732	1733
	1931	2212	2221	2222	2223	2229	2330	2342	2349
	2391	2451	2511	2512	2539	2542	2543	2592	2593
	2710	2813	2815	2822	2823	2832	2833	2861	2866
S40-S49	2869	2930	2944	2945	2950	3101	3102	3329	3701
	3702	3811	3812	3821	3822	3839	3900	4120	4211
	4213	4221	4222	4223	4291	4292	4299	4312	4313
	4319	4321	4329	4391	4399	4520	4530	4541	4542
	4618	4621	4622	4623	4635	4661	4671	4672	4673
	4674	4679	4687	4721	4722	4731	4732	4741	4742
	4743	4744	4784	4789	4921	4922	4930	5212	5221
	5222	5223	5229	5310	5320	7719	7810	7820	7830

INTERVAL ICD-10

NCEA

	8011	8012	8020	8030	8121	8122	8129	9420	
	0210	0220	0810	1011	1012	1013	1031	1033	1041
	1051	1061	1064	1071	1091	1092	1093	1096	1099
	1122	1311	1321	1354	1411	1412	1510	1531	1532
	1533	1540	1610	1621	1622	1623	1629	1722	1732
	1733	2211	2221	2222	2223	2229	2330	2341	2342
	2391	2511	2512	2539	2542	2543	2592	2593	2710
	2759	2813	2822	2823	2832	2833	2861	2866	2869
S50-S59	2930	2944	2945	2950	3011	3101	3102	3329	3701
	3702	3811	3812	3821	3822	3839	3900	4120	4211
	4213	4221	4222	4223	4291	4292	4299	4312	4313
	4319	4321	4322	4329	4391	4399	4520	4621	4622
	4623	4635	4661	4685	4686	4687	4689	4711	4721
	4722	4741	4742	4743	4744	4784	4789	4921	4923
	4924	4929	4930	5212	5221	5222	5223	5229	5310
	5320	7719	7732	7810	7820	7830	8011	8012	8020
	8030	8121	8122	8129	9420				
	0113	0210	0220	0500	0810	1011	1012	1013	1031
	1033	1041	1042	1051	1052	1061	1062	1063	1064
	1071	1072	1091	1092	1093	1094	1096	1099	1122
	1311	1312	1321	1323	1340	1351	1353	1354	1359
	1411	1412	1510	1529	1531	1532	1533	1540	1610
	1621	1622	1623	1629	1710	1721	1722	1731	1732
	1733	1741	1742	1749	1813	1931	2012	2019	2029
	2061	2063	2091	2092	2123	2211	2212	2219	2221
	2222	2223	2229	2311	2312	2319	2330	2341	2342
	2349	2391	2392	2399	2431	2439	2441	2443	2449
	2451	2452	2511	2512	2513	2521	2522	2531	2532
S60-S69	2539	2541	2542	2543	2550	2591	2592	2593	2599
	2632	2651	2710	2721	2722	2732	2733	2740	2751
	2759	2790	2811	2812	2813	2814	2815	2821	2822
	2823	2824	2825	2829	2831	2832	2833	2840	2852
	2853	2854	2861	2862	2864	2865	2866	2869	2920
	2930	2941	2942	2943	2944	2945	2949	2950	3011
	3012	3032	3091	3092	3099	3101	3102	3103	3104
	3220	3230	3240	3250	3291	3299	3319	3329	3701
	3702	3811	3812	3821	3822	3832	3839	3900	4120
	4211	4213	4221	4222	4223	4291	4292	4299	4312
	4313	4319	4321	4322	4329	4391	4399	4520	4621
	4622	4623	4632	4634	4661	4671	4672	4673	4674

Tratado de Toxicología Ocupacional

INTERVAL ICD-10

NCEA

S70-S79	4679	4681	4682	4685	4686	4687	4689	4711	4721
	4722	4741	4742	4743	4744	4789	4930	5211	5212
	5320	5819	5829	7719	7732	7810	7820	7830	8121
	8122	8129	8423	9420	9529				
	0210	0220	1011	1012	1013	1033	1122	1610	1621
	1622	2330	2391	2511	2512	2539	3101	3329	3701
	3702	3811	3812	3821	3822	3839	3900	4120	4211
	4213	4221	4222	4223	4291	4299	4312	4321	4391
	4399	4520	4530	4541	4542	4618	4687	4731	4732
	4741	4742	4743	4744	4784	4789	4921	4930	5212
	5221	5222	5223	5229	5232	5250	5320	7810	7820
	7830	8011	8012	8020	8030	8121	8122	8129	9420
	0210	0220	0230	0500	0710	0810	0990	1011	1012
	1013	1031	1033	1041	1051	1061	1062	1064	1071
	1072	1092	1096	1099	1122	1321	1351	1354	1411
	1412	1510	1531	1532	1540	1610	1621	1622	1623
	1629	1710	1721	1722	1732	1733	1931	2012	2019
	2029	2073	2091	2211	2219	2222	2312	2320	2330
	2341	2342	2391	2439	2443	2449	2451	2511	2512
	2521	2522	2539	2542	2543	2550	2592	2593	2651
	2710	2812	2813	2815	2821	2822	2823	2831	2832
S80-S89	2833	2840	2852	2854	2861	2862	2864	2865	2866
	2869	2930	2943	2944	2945	2950	3011	3101	3102
	3329	3600	3701	3702	3811	3812	3821	3822	3839
	3900	4120	4211	4213	4221	4222	4223	4291	4292
	4299	4312	4313	4319	4321	4322	4329	4391	4399
	4520	4530	4541	4542	4618	4621	4622	4623	4632
	4635	4636	4637	4639	4661	4671	4672	4673	4674
	4679	4681	4682	4685	4686	4687	4689	4711	4722
	4723	4731	4732	4741	4742	4743	4744	4784	4789
	4912	4921	4922	4923	4924	4929	4930	5211	5212
	5221	5222	5223	5229	5232	5250	5310	5320	7719
	7732	7810	7820	7830	8011	8012	8020	8030	8121
	8122	8129	8423	8424	9420				
	0210	0220	0500	0810	1011	1012	1013	1031	1033
	1041	1051	1061	1062	1064	1071	1072	1092	1093
	1122	1311	1321	1351	1354	1411	1412	1510	1532
	1610	1621	1622	1623	1629	1710	1721	1722	1732
	1733	1931	2029	2091	2219	2221	2222	2312	2330
	2341	2342	2391	2431	2439	2441	2443	2449	2451
S90-S99									

INTERVAL ICD-10

NCEA

	2511	2512	2513	2521	2522	2531	2539	2542	2543
	2592	2593	2710	2722	2815	2822	2831	2832	2833
	2840	2852	2853	2854	2861	2862	2865	2866	2869
	2920	2930	2943	2944	2945	2950	3011	3101	3102
	3329	3600	3701	3702	3811	3812	3821	3822	3839
	3900	4120	4211	4213	4221	4222	4223	4291	4292
	4299	4312	4313	4319	4321	4322	4329	4391	4399
	4621	4622	4623	4661	4681	4682	4685	4686	4687
	4689	4711	4784	4912	4921	4922	4930	5111	5120
	5212	5221	5222	5223	5229	5232	5250	5310	5320
	6423	6431	6550	7719	7732	7810	7820	7830	8011
	8012	8020	8030	8121	8122	8129	8423	8424	8610
	9420								
	0210	0220	0710	0810	0892	0910	1011	1013	1020
	1031	1033	1041	1042	1061	1062	1071	1072	1091
	1092	1093	1122	1220	1311	1312	1321	1351	1352
	1353	1411	1412	1510	1531	1532	1533	1540	1610
	1621	1622	1629	1733	1932	2014	2019	2029	2032
	2091	2211	2221	2223	2229	2312	2320	2330	2341
	2342	2391	2451	2511	2512	2521	2522	2539	2542
	2592	2593	2640	2740	2751	2790	2813	2814	2822
T90-T98	2862	2864	2866	2869	2920	2930	2944	2945	2950
	3091	3092	3101	3102	3600	3701	3702	3811	3812
	3821	3822	3839	3900	4120	4211	4213	4221	4291
	4292	4299	4312	4313	4319	4321	4322	4391	4399
	4635	4661	4681	4682	4687	4721	4741	4743	4744
	4784	4922	4923	4924	4929	4930	5012	5021	5030
	5212	5221	5222	5223	5229	5231	5232	5239	5250
	5310	5320	7719	7732	8011	8012	8020	8030	8121
	8122	9420							

"ANNEX V

**LIST OF ACTIVITIES AND CORRESPONDING DEGREES OF
PREVAILING RISK (AS NATIONAL CLASSIFICATION OF
ECONOMIC ACTIVITIES)**

NCEA 2.0	Description	Aliquot
0111- 3/01	Cultivation of rice	3
0111-	Cultivation of maize	3

Tratado de Toxicologia Ocupacional

NCEA 2.0	Description	Aliquot
3/02		
0111- 3/03	Cultivation of wheat	2
0111- 3/99	Growing of other cereals NES	3
0112- 1/01	Cultivation of herbaceous cotton	3
0112- 1/02	Cultivation of jute	3
0112- 1/99	Cultivation of other fibers farming has not previously specified	3
0113- 0/00	Cultivation of sugar cane	3
0114- 8/00	Tobacco cultivation	3
0115- 6/00	Soybean cultivation	3
0116- 4/01	Growing peanuts	2
0116- 4/02	Sunflower Cultivation	2
0116- 4/03	Cultivation of castor	3
0116- 4/99	Cultivation of other oilseeds farming has not previously specified	3
0119- 9/01	Growing Pineapple	2
0119- 9/02	Growing garlic	2
0119- 9/03	Growing potatoes	3
0119- 9/04	Growing onions	2
0119-	Bean	3

NCEA 2.0	Description	Aliquot
9/05		
0119-9/06	Cassava	3
0119-9/07	Melon cultivation	3
0119-9/08	Cultivation of watermelon	2
0119-9/09	Tomato crop creeping	2
0119-9/99	Cultivation of other crops farming has not previously specified	2
0121-1/01	Horticulture, except strawberry	3
0121-1/02	Strawberry fields	3
0122-9/00	Growing flowers and ornamental plants	3
0131-8/00	Orange crop	3
0132-6/00	Grape Cultivation	3
0133-4/01	Growing Acai	1
0133-4/02	Cultivation of banana	3
0133-4/03	Cashew Cultivation	2
0133-4/04	Cultivation of citrus, orange except	3
0133-4/05	Coconut cultivation-the-Bay	3
0133-4/06	Cultivation of guarana	3
0133-	Cultivation of apple	3

Tratado de Toxicologia Ocupacional

NCEA 2.0	Description	Aliquot
4/07		
0133-4/08	Cultivation of papaya	2
0133-4/09	Passion fruit cultivation	3
0133-4/10	Mango cultivation	3
0133-4/11	Peach	3
0133-4/99	Cultivation of permanent crops of fruit not specified elsewhere	3
0134-2/00	Cultivation of coffee	3
0135-1/00	Cocoa cultivation	3
0139-3/01	Cultivation of tea-da-India	3
0139-3/02	Cultivation of yerba mate	3
0139-3/03	Cultivation of black pepper kingdom	3
0139-3/04	Cultivation of plants for seasoning except black pepper kingdom	3
0139-3/05	Cultivation of oil palm	3
0139-3/06	Rubber trees	3
0139-3/99	Cultivation of other crops permanent crops not specified above	3
0141-5/01	Production of certified seed, except forage for grazing	3
0141-5/02	Production of certified seeds of fodder for grazing training	3
0142-	Seedling production and other forms of plant propagation,	2

NCEA 2.0	Description	Aliquot
3/00	certified	
0151- 2/01	Creation of beef cattle	3
0151- 2/02	Cattle for milk	3
0151- 2/03	Cattle, except for beef and dairy	3
0152- 1/01	Creating bubaline	3
0152- 1/02	Creation of equine	2
0152- 1/03	Creation of donkeys and mules	3
0153- 9/01	Goat rearing	3
0153- 9/02	Sheep, including wool production	3
0154- 7/00	Pig farming	3
0155- 5/01	Chicken-cutting	3
0155- 5/02	Production of newly hatched chicks	3
0155- 5/03	Creation of other poultry, except for cutting	2
0155- 5/04	Poultry, except poultry	2
0155- 5/05	Egg Production	3
0159- 8/01	Apiculture	2
0159- 8/02	Creating pets	3
0159-	Creating escargot	1

Tratado de Toxicologia Ocupacional

NCEA 2.0	Description	Aliquot
8/03		
0159-8/04	Creation of silkworm silk	1
0159-8/99	Raising of other animals NES	2
0161-0/01	Service spraying and agricultural pest control	3
0161-0/02	Service for tree pruning crops	3
0161-0/03	Service of land preparation, cultivation and harvesting	3
0161-0/99	Agricultural Support Activities not specified elsewhere	3
0162-8/01	Service of artificial insemination in animals	2
0162-8/02	Service tosquiamento sheep	3
0162-8/03	Service animal husbandry	3
0162-8/99	Activities in support of livestock not previously specified	3
0163-6/00	Postharvest Activities	3
0170-9/00	Hunting and related services	1
0210-1/01	Cultivation of eucalyptus	3
0210-1/02	Cultivation of black wattle	3
0210-1/03	Cultivation of pine	3
0210-1/04	Cultivation of teak	3
0210-	Cultivation of timber species, except eucalyptus, black	2

NCEA 2.0	Description	Aliquot
1/05	wattle, pine and teak	
0210-1/06	Cultivation of seedlings in nurseries	3
0210-1/07	Timber harvesting in plantation forests	3
0210-1/08	Production of charcoal - planted forest	3
0210-1/09	Production of black wattle bark - planted forest	2
0210-1/99	Production of non-timber products NES in planted forests	3
0220-9/01	Logging in native forests	3
0220-9/02	Production of charcoal - natural forests	2
0220-9/03	Gathering nuts-and-stop in native forests	3
0220-9/04	Collection of latex in native forests	1
0220-9/05	Collection of native forests into palm	3
0220-9/06	Conservation of native forests	3
0220-9/99	Collecting non-timber products NES in native forests	3
0230-6/00	Activities to support production forest	3
0311-6/01	Fishing in saltwater fish	3
0311-6/02	Fishing and shellfish in salt water	3
0311-6/03	Collection of other marine products	3
0311-	Activities to support fishing in saltwater	2

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
6/04		
0312-4/01	Fishing in freshwater fish	2
0312-4/02	Fishing and shellfish in freshwater	1
0312-4/03	Collection of other freshwater aquatic products	1
0312-4/04	Activities to support freshwater fisheries	2
0321-3/01	Raising fish in salt water and brackish	2
0321-3/02	Shrimp farming in salt water and brackish	2
0321-3/03	Creation of oysters and mussels in salt water and brackish	3
0321-3/04	Ornamental fish breeding in salt water and brackish	2
0321-3/05	Activities in support of aquaculture in salt water and brackish	2
0321-3/99	Semicultivos crops and aquaculture in salt water and brackish NES	2
0322-1/01	Raising fish in fresh water	3
0322-1/02	Shrimp farming in freshwater	2
0322-1/03	Creation of oysters and mussels in fresh water	2
0322-1/04	Ornamental fish breeding in freshwater	2
0322-1/05	Frogculture	3
0322-1/06	Establishment of alligator	3
0322-	Activities in support of freshwater aquaculture	2

NCEA 2.0	Description	Aliquot
1/07		
0322- 1/99	Semicultivos crops and aquaculture in freshwater NES	3
0500- 3/01	Coal extraction	3
0500- 3/02	Beneficiation of coal	3
0600- 0/01	Extraction of oil and natural gas	3
0600- 0/02	Extraction and processing of shale	3
0600- 0/03	Extraction and processing of oil sands	3
0710- 3/01	Extraction of iron ore	3
0710- 3/02	Pelletizing, sintering and other processings of iron ore	3
0721- 9/01	Extraction of aluminum ore	3
0721- 9/02	Processing of ore of aluminum	3
0722- 7/01	Extraction of tin ore	3
0722- 7/02	Processing of tin ore	3
0723- 5/01	Extraction of manganese ore	3
0723- 5/02	Beneficiation of manganese ore	3
0724- 3/01	Ore extraction of precious metals	3
0724- 3/02	Ore beneficiation of precious metals	3
0725-	Extraction of radioactive minerals	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
1/00		
0729-4/01	Mineral extraction of niobium and titanium	3
0729-4/02	Extraction of tungsten ore	3
0729-4/03	Extraction of nickel ore	3
0729-4/04	Extraction of copper ore, lead, zinc and other nonferrous metallic minerals NES	3
0729-4/05	Beneficiation of copper ore, lead, zinc and other nonferrous metallic minerals NES	2
0810-0/01	Slate extraction and processing associated	3
0810-0/02	Granite extraction and processing associated	3
0810-0/03	Quarrying and processing associated	2
0810-0/04	Extraction of limestone and dolomite and associated processing	3
0810-0/05	Extraction of gypsum and kaolin	2
0810-0/06	Extraction of sand, gravel or gravel and associated processing	3
0810-0/07	Clay mining and processing associated	3
0810-0/08	Gravel extraction and processing associated	3
0810-0/09	Extraction of basalt and associated processing	3
0810-0/10	Processing of gypsum and kaolin associated extraction	1
0810-0/99	Extraction and stamping stones and other building materials and associated processing	3
0891-	Extraction of minerals for the manufacture of fertilizers	3

NCEA 2.0	Description	Aliquot
6/00	and other chemicals	
0892-4/01	Extraction of sea salt	3
0892-4/02	Extraction of rock salt	3
0892-4/03	Refining and further treatment of salt	3
0893-2/00	Extraction of gems (precious and semiprecious stones)	3
0899-1/01	Extraction of graphite	3
0899-1/02	Extraction of quartz	3
0899-1/03	Extraction of asbestos	3
0899-1/99	Extraction of other non-metallic minerals NES	3
0910-6/00	Activities to support the extraction of oil and natural gas	3
0990-4/01	Activities to support the extraction of iron ore	3
0990-4/02	Activities to support the extraction of nonferrous metallic minerals	3
0990-4/03	Activities to support the extraction of nonmetallic minerals	3
1011-2/01	Refrigerator - cattle slaughter	3
1011-2/02	Refrigerator - slaughter of horses	3
1011-2/03	Refrigerator - slaughter of sheep and goats	3
1011-2/04	Refrigerator - Slaughter bubaline	3
1011-	Slaughterhouse - slaughter of cattle under contract -	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
2/05	except for slaughter pigs	
1012- 1/01	Slaughter of poultry	3
1012- 1/02	Slaughter of small animals	3
1012- 1/03	Refrigerator - pig slaughtering	3
1012- 1/04	Slaughterhouse - slaughter of pigs under contract	3
1013- 9/01	Manufacture of meat products	3
1013- 9/02	Preparation of products of slaughter	3
1020- 1/01	Preservation of fish, crustaceans and molluscs	3
1020- 1/02	Manufacture of canned fish, crustaceans and molluscs	3
1031- 7/00	Manufacture of canned fruit	3
1032- 5/01	Manufacture of canned palm heart	2
1032- 5/99	Manufacture of preserved vegetables and other vegetables, except palm	3
1033- 3/01	Manufacture of concentrated juice of fruits, vegetables and legumes	3
1033- 3/02	Manufacture of fruit juices, vegetables and legumes, but concentrates	3
1041- 4/00	Production of crude vegetable oils, except corn oil	3
1042- 2/00	Manufacture of refined vegetable oils, except corn oil	3
1043- 1/00	Manufacture of margarine and other vegetable fats and oils from non-edible animal	2
1051-	Preparation of milk	3

NCEA 2.0	Description	Aliquot
1/00		
1052-0/00	Manufacture of dairy products	3
1053-8/00	Manufacture of ice cream and other edible ices	2
1061-9/01	Rice processing	3
1061-9/02	Manufacture of rice products	3
1062-7/00	Wheat and fabrication of	3
1063-5/00	Production of cassava flour and derivatives	3
1064-3/00	Manufacture of corn flour and derivatives, except corn oils	3
1065-1/01	Manufacture of starches and vegetables	3
1065-1/02	Manufacture of crude corn oil	3
1065-1/03	Manufacture of refined corn oil	3
1066-0/00	Manufacture of animal feed	3
1069-4/00	Milling and manufacturing of products of vegetable origin NES	3
1071-6/00	Manufacture of raw sugar	3
1072-4/01	Manufacture of refined cane sugar	3
1072-4/02	Manufacture of sugar cereal (dextrose) and beet	3
1081-3/01	Processing of coffee	3
1081-	Roasting and grinding coffee	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
3/02		
1082-1/00	Manufacture of products based on coffee	2
1091-1/00	Manufacture of bakery products	3
1092-9/00	Manufacturing cookies and crackers	3
1093-7/01	Manufacture of cocoa products and chocolate	3
1093-7/02	Manufacture of candied fruit, candy and similar	3
1094-5/00	Manufacture of pasta	3
1095-3/00	Manufacture of spices, sauces, seasonings and condiments	3
1096-1/00	Manufacture of food and ready meals	3
1099-6/01	Manufacture of vinegar	3
1099-6/02	Manufacture of food powders	2
1099-6/03	Manufacture of yeast and yeast	1
1099-6/04	Manufacture of ordinary ice	3
1099-6/05	Manufacture of products for infusion (tea, mate, etc.).	3
1099-6/06	Manufacture of natural and artificial sweeteners	3
1099-6/99	Manufacture of other food products NES	3
1111-9/01	Manufacture of cane-sugar	3
1111-	Manufacture of other spirits and distilled beverages	3

NCEA 2.0	Description	Aliquot
9/02		
1112-7/00	Manufacture of Wine	3
1113-5/01	Manufacture of malt whiskey malt including	3
1113-5/02	Manufacture of beer and draft beer	3
1121-6/00	Manufacture of bottled waters	3
1122-4/01	Manufacture of soft drinks	3
1122-4/02	Manufacture of mate tea and other teas ready for consumption	3
1122-4/03	Manufacture of soft drinks, syrups and powders for drinks, soft drinks except fruit	3
1122-4/99	Manufacture of other non-alcoholic beverages not specified above	3
1210-7/00	Industrial processing of tobacco	3
1220-4/01	Manufacture of cigarettes	2
1220-4/02	Manufacture of cigars and cigarillos	3
1220-4/03	Manufacture of cigarette filters	3
1220-4/99	Manufacture of other tobacco products except cigarettes, cigars and cigarillos	3
1311-1/00	Preparation and spinning of cotton fibers	3
1312-0/00	Preparation and spinning of natural textiles, except cotton	3
1313-8/00	Spinning of man-made fibers	3
1314-	Manufacture of sewing and embroidery	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
6/00		
1321-9/00	Weaving of cotton yarn	3
1322-7/00	Weaving yarns of natural fibers, except cotton	3
1323-5/00	Weaving yarns of man-made fibers	3
1330-8/00	Manufacture of knitted fabrics	3
1340-5/01	Stamping and texturing of yarns, fabrics, textile articles and items of clothing	3
1340-5/02	Bleaching, dyeing, twisting into yarn, fabrics, textile articles and items of clothing	3
1340-5/99	Other finishing services of yarns, fabrics, textile articles and items of clothing	3
1351-1/00	Manufacture of textile articles for household use	3
1352-9/00	Manufacture of articles of tapestry	3
1353-7/00	Manufacture of articles of cordage	3
1354-5/00	Manufacture of specialty fabrics, including artifacts	3
1359-6/00	Manufacture of other textiles NES	3
1411-8/01	Manufacture of underwear	3
1411-8/02	Faction underwear	1
1412-6/01	Manufacture of clothing except underwear and tailor-made	3
1412-6/02	Cook, sized, pieces of clothing except underwear	2
1412-	Faction items of clothing except underwear	3

NCEA 2.0	Description	Aliquot
6/03		
1413-4/01	Manufacture of professional clothes, except custom	2
1413-4/02	Clothing, custom, professional clothing	2
1413-4/03	Faction professional clothing	2
1414-2/00	Manufacture of garment accessories, except for security and safety	3
1421-5/00	Manufacture of socks	3
1422-3/00	Manufacture of wearing apparel, knitwear and produced in tricotagens, except socks	3
1510-6/00	Tanning and dressing of leather	3
1521-1/00	Manufacture of travel accessories, handbags and the like of any material	2
1529-7/00	Manufacture of leather NES	3
1531-9/01	Manufacture of leather footwear	2
1531-9/02	Finishing of leather footwear under contract	3
1532-7/00	Manufacture of tennis of any material	2
1533-5/00	Manufacture of footwear made of synthetic material	2
1539-4/00	Shoemaking materials NES	3
1540-8/00	Manufacture of parts for footwear, of any material	3
1610-2/01	Sawmills with split wood	3
1610-	Sawmills without splitting wood	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
2/02		
1621-8/00	Manufacture of laminated wood and sheets of plywood, pressed and sintered	3
1622-6/01	Manufacture of wooden houses, prefabricated	3
1622-6/02	Manufacture of wooden frames and wooden parts for industrial and commercial	3
1622-6/99	Manufacture of other joinery construction	3
1623-4/00	Manufacture of articles of cooperage and wood packaging	3
1629-3/01	Manufacture of various artifacts of wood, except furniture	3
1629-3/02	Manufacture of articles of various cork, bamboo, straw, rattan and other woven materials, except furniture	1
1710-9/00	Cellulose and other folders for papermaking	3
1721-4/00	Papermaking	3
1722-2/00	Manufacture of paperboard and paper-board	3
1731-1/00	Manufacture of paper packaging	3
1732-0/00	Manufacture of cardboard packaging and cardboard	3
1733-8/00	Manufacture of plastic plates and corrugated packaging	3
1741-9/01	Manufacture of continuous forms	2
1741-9/02	Manufacture of paper, cardboard, paper board and corrugated board for commercial and office	3
1742-7/01	Manufacture of disposable diapers	3
1742-	Manufacture of sanitary napkins	3

NCEA 2.0	Description	Aliquot
7/02		
1742-7/99	Manufacture of paper products for household and sanitary-hygiene NES	3
1749-4/00	Manufacture of cellulose pulp, paper, cardboard, cardboard and corrugated cardboard NES	3
1811-3/01	Printing of newspapers	3
1811-3/02	Printing books, magazines and other periodicals	3
1812-1/00	Printing material safety	2
1813-0/01	Printing material for use in advertising	3
1813-0/99	Printing material for other uses	2
1821-1/00	Prepress Services	3
1822-9/00	Graphic finishing services	2
1830-0/01	Sound reproduction in any medium	2
1830-0/02	Video playback on any media	2
1830-0/03	Software reproduction in any medium	1
1910-1/00	Coke Plants	3
1921-7/00	Manufacture of products of petroleum refining	3
1922-5/01	Fuel Formulation	3
1922-5/02	Re-refining lubricating oils	3
1922-	Manufacture of other petroleum products, except products	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
5/99	of the refining	
1931- 4/00	Manufacture of alcohol	3
1932- 2/00	Biofuel production, except alcohol	3
2011- 8/00	Manufacture of chlor-alkali	2
2012- 6/00	Manufacture of intermediates for fertilizers	3
2013- 4/00	Manufacture of fertilizers	2
2014- 2/00	Manufacture of industrial gases	2
2019- 3/01	Preparation of nuclear fuel	3
2019- 3/99	Manufacture of other inorganic chemicals NES	2
2021- 5/00	Manufacture of basic petrochemical products	3
2022- 3/00	Manufacture of intermediates for plasticizers, resins and fibers	3
2029- 1/00	Manufacture of organic chemicals NES	2
2031- 2/00	Manufacture of thermoplastic resins	3
2032- 1/00	Manufacture of thermosetting resins	2
2033- 9/00	Production of elastomers	3
2040- 1/00	Manufacture of man-made fibers	3
2051- 7/00	Manufacture of pesticides	3
2052-	Manufacture of disinfectants bleach	2

NCEA 2.0	Description	Aliquot
5/00		
2061-4/00	Manufacture of soaps and synthetic detergents	3
2062-2/00	Manufacture of cleaning and polishing	3
2063-1/00	Manufacture of cosmetics, perfumes and toiletries	3
2071-1/00	Manufacture of paints, varnishes, enamels and lacquers	3
2072-0/00	Manufacture of printing inks	3
2073-8/00	Manufacture of waterproofing materials, solvents and related products	3
2091-6/00	Manufacture of adhesives and sealants	3
2092-4/01	Manufacture of powders, explosives and detonating	3
2092-4/02	Manufacture of fireworks	2
2092-4/03	Manufacture of Safety Matches	3
2093-2/00	Manufacture of additives for industrial use	3
2094-1/00	Manufacture of catalysts	1
2099-1/01	Manufacture of plastic plates, film, paper and other materials and photographic chemicals	2
2099-1/99	Manufacture of other chemicals NES	3
2110-6/00	Manufacture of pharmaceutical chemicals	3
2121-1/01	Manufacture of allopathic medicines for human use	3
2121-	Manufacture of homeopathic medicines for human use	2

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
1/02		
2121- 1/03	Manufacture of herbal medicines for human use	2
2122- 0/00	Manufacture of veterinary medicinal products	3
2123- 8/00	Manufacture of pharmaceutical preparations	1
2211- 1/00	Manufacture of tires and inner-air	3
2212- 9/00	Reform of used tires	3
2219- 6/00	Manufacture of rubber products NES	3
2221- 8/00	Manufacture of flat rolled and tubular plastic	3
2222- 6/00	Manufacture of plastic containers	3
2223- 4/00	Manufacture of pipes and plastic material for use in construction	3
2229- 3/01	Manufacture of plastic material for personal and household	3
2229- 3/02	Manufacture of plastic material for industrial uses	3
2229- 3/03	Manufacture of plastic material for use in construction, except pipe and fittings	3
2229- 3/99	Manufacture of plastic material for other uses NES	3
2311- 7/00	Manufacture of flat glass and safety	3
2312- 5/00	Manufacture of glass packaging	3
2319- 2/00	Manufacture of glass	3
2320-	Cement manufacturing	3

NCEA 2.0	Description	Aliquot
6/00		
2330-3/01	Manufacture of structural precast concrete in series and custom	3
2330-3/02	Manufacture of articles of cement for use in construction	3
2330-3/03	Manufacture of articles of cement for use in construction	2
2330-3/04	Manufacture of prefabricated houses of concrete	3
2330-3/05	Preparation of mass concrete and mortar construction	3
2330-3/99	Manufacture of other products and concrete products, cement, asbestos, gypsum and similar materials	3
2341-9/00	Manufacture of refractory ceramic products	3
2342-7/01	Manufacture of tiles and flooring	3
2342-7/02	Manufacture of articles of pottery and earthenware for use in construction, except tile floors	3
2349-4/01	Manufacture of ceramic sanitary	3
2349-4/99	Manufacture of refractory ceramic products, NES	3
2391-5/01	Stamping stones, except associated with the extraction	3
2391-5/02	Rigging of stones for construction, except associated with extraction	3
2391-5/03	Rigging plates and execution of works in marble, granite, slate and other stones	3
2392-3/00	Manufacture of lime and gypsum	3
2399-1/01	Decoration, stoning, burning, glazing and other works of pottery, china, glass and crystal	3
2399-	Manufacture of other non-metallic minerals NES	3

Tratado de Toxicologia Ocupacional

NCEA 2.0	Description	Aliquot
1/99		
2411- 3/00	Production of pig iron	3
2412- 1/00	Production of ferroalloys	3
2421- 1/00	Production of semi-finished steel	1
2422- 9/01	Production of flat-rolled carbon steel, coated or not	3
2422- 9/02	Production of rolled special steel	2
2423- 7/01	Production of seamless steel tubes	3
2423- 7/02	Production of long rolled steel, tubes except	2
2424- 5/01	Production of steel wires	2
2424- 5/02	Production of laminated, drawn and profiled steel, except wire	3
2431- 8/00	Production of seamless steel tubes	3
2439- 3/00	Production of iron pipes and other steel	3
2441- 5/01	Production of aluminum and its alloys in primary forms	2
2441- 5/02	Production of rolled aluminum	3
2442- 3/00	Metallurgy of precious metals	2
2443- 1/00	Copper metallurgy	2
2449- 1/01	Zinc production in primary forms	3
2449-	Production of rolled zinc	3

NCEA 2.0	Description	Aliquot
1/02		
2449-1/03	Production of solder and anodes for electroplating	3
2449-1/99	Metallurgy of other non-ferrous metals and alloys NES	3
2451-2/00	Casting of iron and steel	3
2452-1/00	Casting of non-ferrous metals and their alloys	3
2511-0/00	Manufacture of metal structures	3
2512-8/00	Manufacture of metal casings	3
2513-6/00	Manufacture of heavy boiler works	3
2521-7/00	Manufacture of tanks, reservoirs and metallic central heating boilers	3
2522-5/00	Manufacture of steam boilers, except for central heating and vehicles	3
2531-4/01	Production of forged steel	3
2531-4/02	Production of forged non-ferrous metals and their alloys	3
2532-2/01	Production of stamped metal artifacts	3
2532-2/02	Powder metallurgy	3
2539-0/00	Machining services, welding, metal treatment and coating	3
2541-1/00	Manufacture of cutlery	3
2542-0/00	Manufacture of metal works, except frames	3
2543-	Manufacture of tools	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
8/00		
2550-1/01	Manufacture of heavy military equipment, except military fighting vehicles	3
2550-1/02	Manufacture of firearms and ammunition	3
2591-8/00	Manufacture of metal packaging	3
2592-6/01	Manufacture of extruded metal standard	3
2592-6/02	Manufacture of extruded metal, except standard	3
2593-4/00	Manufacture of metal articles for household and personal	3
2599-3/01	Services of manufacture of metal structures for construction	2
2599-3/99	Manufacture of other metal products NES	3
2610-8/00	Manufacture of electronic components	3
2621-3/00	Manufacture of computer equipment	2
2622-1/00	Manufacture of computer peripherals for computer equipment	2
2631-1/00	Manufacture of communication transmitters, parts and accessories	3
2632-9/00	Manufacture of handsets and other communications equipment, parts and accessories	3
2640-0/00	Manufacture of reception, playback, recording and amplification of audio and video	3
2651-5/00	Manufacture of instruments and measurement equipment, test and control	2
2652-3/00	Manufacture of watches and clocks	2
2660-	Manufacture of electrical medical devices and equipment	2

NCEA 2.0	Description	Aliquot
4/00	and irradiation electrotherapies	
2670-1/01	Manufacture of optical equipment and instruments, parts and accessories	2
2670-1/02	Manufacture of photographic and cinematographic apparatus, parts and accessories	3
2680-9/00	Manufacture of blank media, magnetic and optical	3
2710-4/01	Manufacture of generating direct and alternating current, parts and accessories	3
2710-4/02	Manufacture of transformers, inductors, converters, synchronizers and the like, parts and accessories	3
2710-4/03	Manufacture of electric motors, parts and accessories	3
2721-0/00	Manufacture of batteries and electric accumulators except for motor vehicles	3
2722-8/01	Manufacture of batteries and accumulators for automotive vehicles	3
2722-8/02	Reconditioning of batteries and accumulators for automotive vehicles	3
2731-7/00	Manufacture of instruments and equipment for distribution and control of electric power	3
2732-5/00	Manufacture of electrical equipment for circuit installations consumption	3
2733-3/00	Manufacture of wires, cables and electrical conductors insulated	3
2740-6/01	Manufacture of lighting	3
2740-6/02	Manufacture of lamps and other lighting equipment	3
2751-1/00	Manufacture of stoves, refrigerators and washing machines and dryers for household use, parts and accessories	3
2759-7/01	Manufacture of electrical appliances for personal use, parts and accessories	3

NCEA 2.0	Description	Aliquot
2759-7/99	Manufacture of other appliances NES, parts and accessories	3
2790-2/01	Manufacture of electrodes, contacts and other articles of graphite and coal use for electric insulators, and electromagnets	3
2790-2/02	Manufacture of equipment for signaling and alarm	3
2790-2/99	Manufacture of other electrical equipment and appliances NES	2
2811-9/00	Manufacture of engines and turbines, parts and accessories, except for aircraft and road vehicles	2
2812-7/00	Manufacture of hydraulic and pneumatic equipment, parts and accessories, except valves	3
2813-5/00	Manufacture of valves, registers and similar devices, parts and accessories	3
2814-3/01	Manufacture of compressors for industrial use, parts and accessories	3
2814-3/02	Manufacture of compressors for non-industrial parts and accessories	3
2815-1/01	Manufacture of bearings for industrial	2
2815-1/02	Manufacture of transmission equipment for industrial purposes, except bearings	3
2821-6/01	Manufacture of industrial furnaces, appliances and non-electric to thermal plants, parts and accessories	3
2821-6/02	Manufacture of ovens and electric ovens for industrial purposes, parts and accessories	3
2822-4/01	Manufacture of machinery, equipment and apparatus for lifting and transporting of people, parts and accessories	3
2822-4/02	Manufacture of machinery, equipment and apparatus for transporting and lifting loads, parts and accessories	3
2823-2/00	Manufacture of electrical machinery and apparatus for cooling and ventilation systems for industrial and commercial use, parts and accessories	3
2824-	Manufacture of appliances and air conditioning	2

NCEA 2.0	Description	Aliquot
1/01	equipment for industrial use	
2824-1/02	Manufacture of appliances and air conditioning equipment for non-industrial	2
2825-9/00	Manufacture of machinery and equipment for sanitation and environmental protection, parts and accessories	2
2829-1/01	Manufacture of typewriters, calculators and other non-electronic office, parts and accessories	2
2829-1/99	Manufacture of machinery and equipment commonly used NES, parts and accessories	3
2831-3/00	Manufacture of agricultural tractors, parts and accessories	3
2832-1/00	Manufacture of agricultural irrigation equipment, parts and accessories	3
2833-0/00	Manufacture of machinery and equipment for agriculture and livestock, parts and accessories, except for irrigation	3
2840-2/00	Manufacture of machine tools, parts and accessories	3
2851-8/00	Manufacture of machinery and equipment for mining and oil extraction, parts and accessories	3
2852-6/00	Manufacture of machinery and equipment for use in mining, parts and accessories, except the extraction of petroleum	3
2853-4/00	Manufacture of tractors, parts and accessories, except agricultural	3
2854-2/00	Manufacture of machinery and equipment for earthmoving, paving and construction, parts and accessories, except tractors	3
2861-5/00	Manufacture of machinery for the metalworking industry, parts and accessories, except machine tools	3
2862-3/00	Manufacture of machinery and equipment for the food, beverages and tobacco, parts and accessories	3
2863-1/00	Manufacture of machinery and equipment for the textile industry, parts and accessories	3
2864-0/00	Manufacture of machinery and equipment for the industries of clothing, leather and footwear, parts and	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
	accessories	
2865-8/00	Manufacture of machinery and equipment for the manufacture of pulp, paper and paperboard and articles, parts and accessories	3
2866-6/00	Manufacture of machinery and equipment for the plastics industry, parts and accessories	3
2869-1/00	Manufacture of machinery and equipment for specific industrial use NES, parts and accessories	3
2910-7/01	Manufacture of cars, vans and utilities	3
2910-7/02	Manufacture of chassis with engines for cars, vans and utilities	3
2910-7/03	Manufacture of engines for cars, vans and Utilities	3
2920-4/01	Manufacture of trucks and buses	3
2920-4/02	Manufacture of engines for trucks and buses	2
2930-1/01	Manufacture of cabs, truck bodies and trailers	3
2930-1/02	Manufacture of bodies for buses	3
2930-1/03	Manufacture of cabs, truck bodies and trailers for other vehicles, except trucks and buses	3
2941-7/00	Manufacture of parts and accessories for motor system of automotive vehicles	3
2942-5/00	Manufacture of parts and accessories for the march and transmission systems of automotive vehicles	3
2943-3/00	Manufacture of parts and accessories for the brake system of automotive vehicles	3
2944-1/00	Manufacture of parts and accessories for the steering system and suspension of automotive vehicles	3
2945-0/00	Manufacture of electrical and electronic equipment for motor vehicles, except batteries	3

NCEA 2.0	Description	Aliquot
2949-2/01	Manufacture of seats and upholstery for automotive vehicles	3
2949-2/99	Manufacture of parts and accessories for motor vehicles not specified elsewhere	3
2950-6/00	Recovery and reconditioning of engines for automotive vehicles	3
3011-3/01	Construction of large vessels	3
3011-3/02	Construction of vessels for commercial and special purpose except large	3
3012-1/00	Construction of boats for sport and leisure	3
3031-8/00	Manufacture of locomotives, wagons and other materials undercarriages	3
3032-6/00	Manufacture of parts and accessories for rail vehicles	3
3041-5/00	Manufacture of aircraft	2
3042-3/00	Manufacture of turbines, engines and other components and parts for aircraft	2
3050-4/00	Manufacture of military fighting vehicles	2
3091-1/00	Manufacture of motorcycles, parts and accessories	3
3092-0/00	Manufacture of bicycles and non-motorized tricycles, parts and accessories	3
3099-7/00	Manufacture of transport equipment NES	3
3101-2/00	Manufacture of wooden furniture with a predominance	3
3102-1/00	Manufacture of furniture with a predominance of metal	3
3103-9/00	Manufacture of furniture of other materials, except wood and metal	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
3104-7/00	Manufacture of mattresses	3
3211-6/01	Lapidary gem	2
3211-6/02	Manufacture of articles of jewelry and metalwork	2
3211-6/03	Striking of coins and medals	2
3212-4/00	Manufacture of jewelry and similar articles	3
3220-5/00	Manufacture of musical instruments, parts and accessories	3
3230-2/00	Manufacture of articles for fishing and sport	3
3240-0/01	Manufacture of electronic games	2
3240-0/02	Manufacture of billiard tables, billiard accessories and not associated with the lease	2
3240-0/03	Manufacture of billiard tables, billiard accessories and associated lease	2
3240-0/99	Manufacture of toys and other recreational games NES	3
3250-7/01	Manufacture of non-electronics and appliances for medical, surgical, dental and laboratory	2
3250-7/02	Manufacture of furniture for medical, surgical, dental and laboratory	3
3250-7/03	Manufacture of instruments and apparatus to correct physical defects in general and orthopedic custom	2
3250-7/04	Manufacture of instruments and apparatus to correct physical defects and orthotic devices in general, except custom	2
3250-7/05	Manufacture of materials for medicine and dentistry	3
3250-7/06	Dental services	2

NCEA 2.0	Description	Aliquot
3250-7/07	Manufacture of optical	3
3250-7/08	Manufacture of articles of nonwoven fabric for use in dental-medical-hospital	2
3291-4/00	Manufacture of brushes, mops and brooms	3
3292-2/01	Manufacture of protective clothing and safety and fire-resistant	3
3292-2/02	Manufacturing equipment and accessories for personal and professional security	3
3299-0/01	Manufacture of umbrellas and similar	2
3299-0/02	Manufacture of pens, pencils and other office supplies	2
3299-0/03	Manufacture of letters, posters and cards of any material, except bright	2
3299-0/04	Manufacture of illuminated signs and panels	3
3299-0/05	Manufacture of sewing trims	3
3299-0/99	Manufacture of miscellaneous products NES	3
3311-2/00	Maintenance and repair of tanks, metal vessels and boilers, except for vehicles	3
3312-1/01	Maintenance and repair of communication equipment transmitters	2
3312-1/02	Maintenance and repair of equipment and measuring instruments, test and control	2
3312-1/03	Maintenance and repair of medical electrical apparatus and equipment and irradiation electrotherapies	1
3312-1/04	Maintenance and repair of equipment and optical instruments	3
3313-9/01	Maintenance and repair of generators, transformers and electric motors	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
3313-9/02	Maintenance and repair of electric accumulators except for vehicles	2
3313-9/99	Maintenance and repair of machinery, appliances and equipment NES	3
3314-7/01	Maintenance and repair of non-electric motors	1
3314-7/02	Maintenance and repair of hydraulic and pneumatic equipment, except valves	3
3314-7/03	Maintenance and repair of industrial valves	2
3314-7/04	Maintenance and repair of compressors	3
3314-7/05	Maintenance and repair of transmission equipment for industrial	2
3314-7/06	Maintenance and repair of machinery, apparatus and equipment for thermal plants	3
3314-7/07	Maintenance and repair of machinery and equipment and ventilation systems for industrial and commercial	3
3314-7/08	Maintenance and repair of machinery, equipment and apparatus for transporting and lifting loads	3
3314-7/09	Maintenance and repair of typewriters, calculators and other non-electronic office	3
3314-7/10	Maintenance and repair of machinery and equipment for general use NES	3
3314-7/11	Maintenance and repair of machinery and equipment for agriculture and livestock	3
3314-7/12	Maintenance and repair of agricultural tractors	3
3314-7/13	Maintenance and repair of machine tools	3
3314-7/14	Maintenance and repair of machinery and equipment for mining and oil extraction	3
3314-7/15	Maintenance and repair of machinery and equipment for use in mining, except oil extraction	2

NCEA 2.0	Description	Aliquot
3314-7/16	Maintenance and repair of machinery, except agricultural	3
3314-7/17	Maintenance and repair of machinery and equipment, earthmoving, paving and construction, except tractors	3
3314-7/18	Maintenance and repair of machinery for the metalworking industry, except machine tools	3
3314-7/19	Maintenance and repair of machinery and equipment for the food, beverages and tobacco	3
3314-7/20	Maintenance and repair of machinery and equipment Textile, apparel, leather and footwear	2
3314-7/21	Maintenance and repair of machinery for the pulp, paper and paperboard and articles	3
3314-7/22	Maintenance and repair of machinery for the plastics industry	3
3314-7/99	Maintenance and repair of machinery and equipment for industrial uses NES	3
3315-5/00	Maintenance and repair of rail vehicles	3
3316-3/01	Maintenance and repair of aircraft, except the maintenance on the track	2
3316-3/02	Maintenance of aircraft on the runway	1
3317-1/01	Maintenance and repair of ships and floating structures	3
3317-1/02	Maintenance and repair of vessels for sport and leisure	2
3319-8/00	Maintenance and repair of equipment and products NES	3
3321-0/00	Installation of industrial machinery and equipment	3
3329-5/01	Assembly services of any piece of furniture	3
3329-5/99	Installation of other equipment NES	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
3511-5/00	Electric power generation	3
3512-3/00	Transmission of electricity	3
3513-1/00	Wholesale electricity	1
3514-0/00	Electric power distribution	3
3520-4/01	Gas production, natural gas processing	2
3520-4/02	Distribution of gaseous fuels for urban networks	2
3530-1/00	Production and distribution of steam, hot water and air conditioning	2
3600-6/01	Collection, purification and distribution of water	3
3600-6/02	Water distribution by trucks	2
3701-1/00	Management of sewage	3
3702-9/00	Activities related to sewage, except for network management	3
3811-4/00	Collection of non-hazardous waste	3
3812-2/00	Collection of hazardous waste	2
3821-1/00	Treatment and disposal of non-hazardous waste	3
3822-0/00	Treatment and disposal of hazardous waste	3
3831-9/01	Recovery of scrap aluminum	3
3831-9/99	Recovery of metallic materials, except aluminum	3

NCEA 2.0	Description	Aliquot
3832-7/00	Recovery of plastics	3
3839-4/01	Composting plants	3
3839-4/99	Recovery of materials NES	3
3900-5/00	Decontamination and other waste management services	2
4110-7/00	Real estate developments	3
4120-4/00	Construction of buildings	3
4211-1/01	Construction of roads and railways	3
4211-1/02	Painting for signage in bus stations and airports runways	3
4212-0/00	Construction of special works of art	3
4213-8/00	Urbanization works - streets, squares and sidewalks	3
4221-9/01	Construction of dams and reservoirs for power generation	3
4221-9/02	Construction of stations and distribution networks for electricity	3
4221-9/03	Maintenance of distribution networks for electricity	3
4221-9/04	Construction of stations and telecommunications networks	3
4221-9/05	Maintenance of stations and telecommunications networks	3
4222-7/01	Construction of water supply networks, sewage and construction related, except irrigation works	3
4222-7/02	Irrigation works	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
4223-5/00	Construction of pipelines for transport networks, except for water and sewer	3
4291-0/00	Harbors, maritime and river	3
4292-8/01	Assembly of metal structures	3
4292-8/02	Works of industrial assembly	3
4299-5/01	Construction of sports and recreation facilities	3
4299-5/99	Other civil engineering works not previously specified	3
4311-8/01	Demolition of buildings and other structures	3
4311-8/02	Preparation of plots and land clearance	3
4312-6/00	Drilling and boring	3
4313-4/00	Earthwork	3
4319-3/00	Land preparation services NES	2
4321-5/00	Electrical installation and maintenance	3
4322-3/01	Plumbing, sanitary and gas	3
4322-3/02	Installation and maintenance of central air conditioning, ventilation and cooling	3
4322-3/03	Facilities of the fire prevention system	3
4329-1/01	Installation of hoardings	2
4329-1/02	Installation of equipment for guidance in shipping lake and river	2

NCEA 2.0	Description	Aliquot
4329-1/03	Installation, maintenance and repair of elevators, escalators and moving walkways, except manufacturing itself	2
4329-1/04	Assembly and installation of systems and equipment, lighting and signs on public roads, ports and airports	3
4329-1/05	Thermal treatments, acoustical or vibration	3
4329-1/99	Other construction installation works not previously specified	3
4330-4/01	Waterproofing in civil engineering works	3
4330-4/02	Installation of doors, windows, ceilings, walls and closets of any material	3
4330-4/03	Finishing works in plaster and stucco	3
4330-4/04	Painting services for buildings in general	3
4330-4/05	Application of coatings and resins for indoor and outdoor	3
4330-4/99	Other articles of completion	3
4391-6/00	Foundation work	3
4399-1/01	Administration works	3
4399-1/02	Erection and dismantling of scaffolding and other temporary structures	3
4399-1/03	Bricklaying	3
4399-1/04	Operation and provision of equipment to lift and transport people and cargo for use in works	3
4399-1/05	Drilling and construction of water wells	3
4399-1/99	Specialized services for construction NES	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
4511-1/01	Retail sales of cars, new trucks and utility	2
4511-1/02	Retail sales of cars, vans and utilities	3
4511-1/03	Wholesale trade of cars, trucks and utility of new and used	2
4511-1/04	Wholesale trade of new and used trucks	2
4511-1/05	Wholesale trade of trailers and semi-trailers of new and used	3
4511-1/06	Wholesale trade of buses and minibuses used and new	1
4512-9/01	Trade representatives and agents of the trade of automotive vehicles	2
4512-9/02	Trade under the assignment of motor vehicles	3
4520-0/01	Maintenance and mechanical repair of automotive vehicles	3
4520-0/02	Services body shop or auto body and painting of automotive vehicles	3
4520-0/03	Maintenance and repair of electric motor vehicles	3
4520-0/04	Alignment and balancing services for automotive vehicles	2
4520-0/05	Washing services, lubrication and polishing of motor vehicles	3
4520-0/06	Tire repair services for automotive vehicles	3
4520-0/07	Installation services, maintenance and repair of accessories for automotive vehicles	3
4530-7/01	Wholesale trade of parts and accessories for new automobiles	2
4530-7/02	Wholesale trade of tires and inner-tubes	2

NCEA 2.0	Description	Aliquot
4530-7/03	Retail sales of parts and accessories for new automobiles	2
4530-7/04	Retail sales of used parts and accessories for motor vehicles	2
4530-7/05	Retail sales of tires and inner-tubes	2
4530-7/06	Trade representatives and agents of parts and accessories new and used automotive vehicles	2
4541-2/01	Wholesale trade of motorcycles and scooters	2
4541-2/02	Wholesale trade of parts and accessories for motorcycles and scooters	3
4541-2/03	Retail sales of new motorcycles and scooters	3
4541-2/04	Retail sales of motorcycles and scooters used	3
4541-2/05	Retail sales of parts and accessories for motorcycles and scooters	3
4542-1/01	Trade representatives and commission trade of motorcycles and scooters, parts and accessories	1
4542-1/02	Trade in motorcycles and scooters assignment	2
4543-9/00	Maintenance and repair of motorcycles and scooters	2
4611-7/00	Trade representatives and agents of trade in agricultural raw materials and live animals	3
4612-5/00	Trade representatives and agents of trade in fuels, minerals, steel products and chemicals	2
4613-3/00	Trade representatives and agents of trade in timber, building material and hardware	3
4614-1/00	Trade representatives and agents of trade in machinery, equipment, ships and aircraft	2
4615-0/00	Trade representatives and trade agents to appliances, furniture and household articles	2

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
4616-8/00	Trade representatives and agents of trade in textiles, clothing, footwear and travel goods	1
4617-6/00	Trade representatives and agents of trade in food products, beverages and tobacco	3
4618-4/01	Trade representatives and agents of trade in medicines, cosmetics and perfumery	2
4618-4/02	Trade representatives and agents of trade tools and materials, medical and dental hospital	2
4618-4/03	Trade representatives and agents of trade journals, magazines and other publications	3
4618-4/99	Other business representatives and trade agents specializing in products NES	2
4619-2/00	Trade representatives and agents of trade in goods in general non-specialist	2
4621-4/00	Wholesale coffee beans	3
4622-2/00	Wholesale Soy	3
4623-1/01	Wholesale of live animals	3
4623-1/02	Wholesale of hides, wool, skins and other non-edible products of animal origin	3
4623-1/03	Wholesale cotton	2
4623-1/04	Wholesale trade of tobacco leaf not benefited	3
4623-1/05	Wholesale Cocoa	2
4623-1/06	Wholesale trade of seeds, flowers, plants and grasses	3
4623-1/07	Wholesale Sisal	2
4623-1/08	Wholesale of agricultural raw materials with activity associated fractionation and packaging	3

NCEA 2.0	Description	Aliquot
4623-1/09	Wholesale feed	3
4623-1/99	Wholesale of agricultural raw materials, not specified elsewhere	3
4631-1/00	Wholesale milk and dairy	3
4632-0/01	Wholesale of cereals and legumes benefit	3
4632-0/02	Wholesale flours, starches	3
4632-0/03	Wholesale of cereals and legumes benefit, flours, starches, with activity associated fractionation and packaging	3
4633-8/01	Wholesale trade of fruits, vegetables, roots, tubers, vegetables and fresh vegetables	3
4633-8/02	Wholesale trade of live poultry and eggs	2
4633-8/03	Wholesale rabbits and other small livestock feed	2
4634-6/01	Wholesale trade of beef and pork and derivatives	3
4634-6/02	Wholesale birds slaughtered and derivatives	3
4634-6/03	Wholesale of fish and seafood	3
4634-6/99	Wholesale trade of meat and meat of other animals	2
4635-4/01	Wholesale trade of mineral water	3
4635-4/02	Wholesale of beer, beer and soda	3
4635-4/03	Wholesale trade of beverages and packaging activity of fractionation associated	3
4635-4/99	Wholesale of drinks not specified above	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
4636-2/01	Wholesale Smoke benefited	3
4636-2/02	Wholesale cigarettes, cigars and cigarillos	2
4637-1/01	Trade wholesaler of roasted coffee and soluble	3
4637-1/02	Wholesale Sugar	2
4637-1/03	Wholesale trade of oils and fats	2
4637-1/04	Wholesale trade of bread, cakes, biscuits and similar	2
4637-1/05	Wholesale Pasta	3
4637-1/06	Wholesale ice cream	2
4637-1/07	Wholesale chocolates, sweets, candies, chocolates and similar	3
4637-1/99	Wholesale specializing in other food products NES	3
4639-7/01	Wholesale trade of food products in general	3
4639-7/02	Wholesale trade of food products in general, with activity associated fractionation and packaging	3
4641-9/01	Wholesale fabric	2
4641-9/02	Wholesale of bedding, bath and table	3
4641-9/03	Wholesale of haberdashery articles	3
4642-7/01	Trade wholesaler of apparel and accessories, except professional and security	1
4642-7/02	Wholesale of clothing and accessories for professional and occupational safety	2

NCEA 2.0	Description	Aliquot
4643-5/01	Wholesale shoes	2
4643-5/02	Wholesale of handbags, luggage and travel goods	1
4644-3/01	Wholesale trade of medicines and drugs for human use	2
4644-3/02	Wholesale trade of medicines and drugs for veterinary use	2
4645-1/01	Wholesale of instruments and materials for medical, surgical, hospital and laboratories	1
4645-1/02	Wholesale prosthetic and orthopedic articles	2
4645-1/03	Wholesale trade of dental products	2
4646-0/01	Wholesale of cosmetics and perfumery	2
4646-0/02	Wholesale of toiletries	2
4647-8/01	Wholesale trade of office equipment and stationery	2
4647-8/02	Wholesale of books, newspapers and other publications	3
4649-4/01	Wholesale of electrical equipment for personal and domestic	2
4649-4/02	Wholesale electronics for personal and household	3
4649-4/03	Wholesale of bicycles, tricycles and other recreational vehicles	3
4649-4/04	Wholesale furniture and beddings	3
4649-4/05	Wholesale of upholstery, curtains and blinds	2
4649-4/06	Wholesale of chandeliers, lamps and lampshades	2

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
4649-4/07	Wholesale Movies, CDs, DVDs, tapes and discs	1
4649-4/08	Wholesale cosmetics, household cleaning and maintenance	3
4649-4/09	Wholesale cosmetics, household cleaning and maintenance, with activity associated fractionation and packaging	2
4649-4/10	Wholesale jewelry, watches and jewelry, including precious and semiprecious stones polished	1
4649-4/99	Wholesale of other equipment and articles of personal and household NES	2
4651-6/01	Wholesale computer equipment	1
4651-6/02	Trade wholesaler of office supplies	1
4652-4/00	Wholesale trade of electronics and telephony equipment and communication	1
4661-3/00	Wholesale of machinery, apparatus and equipment for agricultural use, parts and accessories	2
4662-1/00	Wholesale of machinery, equipment for earthmoving, mining and construction and parts	3
4663-0/00	Wholesale trade of machinery and equipment for industrial use, and parts	2
4664-8/00	Wholesale of machinery, apparatus and equipment for use in medical, dental, hospital, and parts	2
4665-6/00	Wholesale trade of machinery and equipment for commercial use, and parts	2
4669-9/01	Wholesale trade of pumps and compressors, and parts	2
4669-9/99	Wholesale of other machinery and equipment NES, and parts	2
4671-1/00	Wholesale trade of timber and timber products	3
4672-9/00	Wholesale of hardware and tools	3

NCEA 2.0	Description	Aliquot
4673-7/00	Wholesale trade of electric material	2
4674-5/00	Wholesale Cement	2
4679-6/01	Wholesale of paints, varnishes and similar	2
4679-6/02	Wholesale Marble & Granite	3
4679-6/03	Wholesale of glass, mirrors and stained glass	3
4679-6/04	Wholesale specialist construction materials NES	2
4679-6/99	Wholesale trade of construction materials in general	3
4681-8/01	Wholesale trade of fuel ethanol, biodiesel, gasoline and other petroleum products, except lubricants, not performed by the carrier retailer (TRR)	3
4681-8/02	Wholesale fuel carried by conveyor retailer (TRR)	3
4681-8/03	Wholesale trade of biomass fuels, except for alcohol fuel	3
4681-8/04	Wholesale trade of mineral fuels, raw	2
4681-8/05	Wholesale of lubricants	2
4682-6/00	Wholesale trade of liquefied petroleum gas (LPG)	3
4683-4/00	Wholesale of pesticides, fertilizers and soil amendments	2
4684-2/01	Wholesale of resins and elastomers	2
4684-2/02	Wholesale of solvents	3
4684-2/99	Wholesale of other petrochemical products NES	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
4685-1/00	Wholesale trade in steel products and steel workers, except construction	3
4686-9/01	Wholesale of paper and cardboard, raw	2
4686-9/02	Wholesale Packaging	3
4687-7/01	Wholesale waste paper and cardboard	3
4687-7/02	Wholesale of waste and scrap non-metallic, except paper and cardboard	3
4687-7/03	Wholesale of waste and scrap metal	3
4689-3/01	Wholesale trade of products of mining, except fuels	2
4689-3/02	Wholesale trade of yarns and fibers benefit	2
4689-3/99	Wholesale specializing in other intermediate products NES	2
4691-5/00	Wholesale general merchandise, with predominance of food products	2
4692-3/00	Wholesale general merchandise, with a predominance of agricultural inputs	2
4693-1/00	Wholesale general merchandise, with no predominance of food or agricultural inputs	2
4711-3/01	Retail sale of general merchandise, with a predominance of food products - hypermarkets	3
4711-3/02	Retail sale of general merchandise, predominantly food - supermarkets	3
4712-1/00	Retail sale of general merchandise, predominantly food - convenience stores, grocery stores and warehouses	2
4713-0/01	Department stores or magazines	3
4713-0/02	Variety stores, except department stores or magazines	2

NCEA 2.0	Description	Aliquot
4713-0/03	Duty free shopping for international airports	2
4721-1/01	Bakery and confectionery with a predominance of own production	3
4721-1/02	Bakery and confectionery predominantly retail	2
4721-1/03	Retail sale of dairy products and cold	2
4721-1/04	Retail sale of sweets, candies, chocolates and similar	3
4722-9/01	Retail sale of meat - butcher	3
4722-9/02	Fishmonger	2
4723-7/00	Retail sale of beverages	3
4724-5/00	Retail sale of fruits and vegetables	3
4729-6/01	Tobacco shop	1
4729-6/99	Retail trade of food products in general or specialized in food products NES	2
4731-8/00	Retail trade of fuels for motor vehicles	3
4732-6/00	Retail sale of lubricants	2
4741-5/00	Retail sale of paints and materials for painting	2
4742-3/00	Retail sale of electrical equipment	3
4743-1/00	Retail Glass	3
4744-0/01	Retail sale of hardware and tools	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
4744-0/02	Retail sale of wood and artifacts	3
4744-0/03	Retail sale of hydraulic materials	2
4744-0/04	Retail sale of lime, sand, gravel, bricks and tiles	3
4744-0/05	Retail building materials NES	3
4744-0/99	Retail sale of construction materials in general	3
4751-2/00	Retail sale of specialized equipment and computer supplies	2
4752-1/00	Retail specialized telephony equipment and communication	2
4753-9/00	Specialized retail trade of household appliances and audio equipment and video	2
4754-7/01	Retail furniture	2
4754-7/02	Retail sale of beddings	2
4754-7/03	Retail sale of lighting equipment	2
4755-5/01	Retail sale of textiles	2
4755-5/02	Retail trade of haberdashery articles	2
4755-5/03	Retail trade of bedding, bath and table	3
4756-3/00	Retail specialized musical instruments and accessories	2
4757-1/00	Specialized retail trade of parts and accessories for consumer electronics, household appliances, except computer and communication	2
4759-8/01	Retail trade of upholstery, curtains and blinds	2

NCEA 2.0	Description	Aliquot
4759-8/99	Retail sale of other articles of personal and household NES	2
4761-0/01	Retail sale of books	1
4761-0/02	Retail sale of newspapers and magazines	1
4761-0/03	Retail sale of stationery	2
4762-8/00	Retail sale of albums, CDs, DVDs and tapes	1
4763-6/01	Retail sale of toys and recreational	2
4763-6/02	Retail sale of sporting goods	1
4763-6/03	Retail sale of bicycles and tricycles, parts and accessories	1
4763-6/04	Retail sale of articles for hunting, fishing and camping	1
4763-6/05	Retail sale of boats and other recreational vehicles, parts and accessories	2
4771-7/01	Retail sale of pharmaceutical products, without manipulation of formulas	2
4771-7/02	Retail sale of pharmaceutical products, the manipulation of formulas	2
4771-7/03	Retail sale of pharmaceutical products Homeopathic	1
4771-7/04	Retail sale of veterinary medicines	3
4772-5/00	Retail sale of cosmetics, perfumes and toiletries	2
4773-3/00	Retail sale of medical and orthopedic	1
4774-1/00	Retail sale of optical articles	2

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
4781-4/00	Retail sale of clothing articles and accessories	2
4782-2/01	Retail sale of footwear	2
4782-2/02	Retail sale of travel	1
4783-1/01	Retail sale of articles of jewelry	1
4783-1/02	Retail sale of clocks and watches	2
4784-9/00	Retail sale of liquefied petroleum gas (LPG)	3
4785-7/01	Retail sale of antiques	2
4785-7/99	Retail sale of other articles used	3
4789-0/01	Retail sale of souvenirs, jewelry and handicrafts	2
4789-0/02	Retail sale of natural plants and flowers	3
4789-0/03	Retail sale of art objects	1
4789-0/04	Retail sale of live animals and food items and pet	3
4789-0/05	Retail sale of household cleaning products products	3
4789-0/06	Retail sale of fireworks and pyrotechnics	2
4789-0/07	Retail sale of office equipment	2
4789-0/08	Retail sale of articles and photographic film	1
4789-0/09	Retail sale of weapons and ammunition	2

NCEA 2.0	Description	Aliquot
4789-0/99	Retail sale of other products NES	2
4911-6/00	Cargo transportation	3
4912-4/01	Intercity passenger rail and interstate	3
4912-4/02	Rail passenger transport in metropolitan and municipal	3
4912-4/03	Transportation subway	3
4921-3/01	Collective passenger transport by road, with a fixed itinerary, municipal	3
4921-3/02	Collective passenger transport by road, with a fixed route, intercity in the metropolitan area	3
4922-1/01	Collective passenger transport by road, with a fixed route, intercity, except in the metropolitan area	3
4922-1/02	Collective passenger transport by road, with a fixed route, interstate	3
4922-1/03	Collective passenger transport by road, with a fixed itinerary, international	3
4923-0/01	Taxi service	3
4923-0/02	Passenger service - car rental with driver	3
4924-8/00	School Transport	3
4929-9/01	Collective passenger transport by road, under a charter, municipal	3
4929-9/02	Collective passenger transport by road, under a charter, intercity, interstate and international	3
4929-9/03	Organization of excursions own road vehicles, municipal	3
4929-9/04	Organization of excursions own road vehicles, intercity, interstate and international	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
4929-9/99	Other road passenger transport NES	2
4930-2/01	Road freight, except products and dangerous changes, municipal	3
4930-2/02	Road freight except hazardous products and changes, intermunicipal, interstate and international	3
4930-2/03	Road transport of dangerous goods	3
4930-2/04	Road transport changes	3
4940-0/00	Pipeline transport	1
4950-7/00	Tourist trains, cable cars and the like	3
5011-4/01	Maritime cabotage - Load	3
5011-4/02	Maritime cabotage - passengers	2
5012-2/01	Long-distance shipping - Cargo	3
5012-2/02	Shipping long haul - Passenger	2
5021-1/01	Inland waterway transport cargo, municipal, except crossing	3
5021-1/02	Inland waterway transport freight, intercity, interstate and internationally, except crossing	3
5022-0/01	Inland waterway transport of passengers in regular lines, municipal, except crossing	2
5022-0/02	Inland waterway transport of passengers in regular lines, intercity, interstate and internationally, except crossing	2
5030-1/01	Maritime support navigation	3
5030-1/02	Port support navigation	1

NCEA 2.0	Description	Aliquot
5091-2/01	Transport by inland crossing, municipal	3
5091-2/02	Transport by inland crossing, inter	3
5099-8/01	Water transportation for tours	1
5099-8/99	Other water transportation NES	1
5111-1/00	Regular passenger air transport	3
5112-9/01	Air taxi service and rental of aircraft with crew	3
5112-9/99	Other services of passenger air transport Non-regular	3
5120-0/00	Air freight	2
5130-7/00	Space transportation	1
5211-7/01	General stores - issuance of warrant	3
5211-7/02	Furniture storage	2
5211-7/99	Stocks of goods to third parties except general stores and furniture	3
5212-5/00	Loading and unloading	3
5221-4/00	Dealers highways, bridges, tunnels and related services	3
5222-2/00	Bus and rail terminals	3
5223-1/00	Parking of vehicles	3
5229-0/01	Support services for transport by taxi, including call centers	1

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
5229-0/02	Towing vehicle	3
5229-0/99	Other activities for land transport not previously specified	3
5231-1/01	Management of port infrastructure	2
5231-1/02	Terminal operations	3
5232-0/00	Activities shipping agency	2
5239-7/00	Activities incidental to water transportation not specified above	3
5240-1/01	Operation of airports and landing fields	2
5240-1/99	Activities incidental to air transportation, except operation of airports and landing fields	3
5250-8/01	Curates of Ordinances	1
5250-8/02	Activities of customs brokers	3
5250-8/03	Cargo management, except for shipping	3
5250-8/04	Organisation of freight transport logistics	3
5250-8/05	Multimodal transport operator - OTM	3
5310-5/01	Activities of the National Post	3
5310-5/02	Activities franchisees and licensees of the National Post	2
5320-2/01	Pouch services not performed by the National Post	3
5320-2/02	Delivery services	3

NCEA 2.0	Description	Aliquot
5510-8/01	Hotels	2
5510-8/02	Apart-hotels	2
5510-8/03	Motels	2
5590-6/01	Hostels, except assistance	3
5590-6/02	Campings	1
5590-6/03	Pension (lodging)	2
5590-6/99	Other lodging NES	2
5611-2/01	Restaurants and similar	2
5611-2/02	Bars and other establishments specialized in serving drinks	3
5611-2/03	Snack bars, tea houses, juice and the like	3
5612-1/00	Services vendors supply	3
5620-1/01	Providing food prepared primarily for businesses	3
5620-1/02	Food services for events and receptions - Catering	2
5620-1/03	Canteens - private food services	3
5620-1/04	Providing food prepared mainly for household consumption	3
5811-5/00	Book publishing	2
5812-3/00	Publishing of newspapers	2

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
5813-1/00	Magazine publishing	3
5819-1/00	Editing entries, lists and other graphic products	2
5821-2/00	Integrated into the print edition of books	2
5822-1/00	Integrated editing of newsprint	2
5823-9/00	Integrated editing to magazine printing	2
5829-8/00	Integrated editing to printing records, lists and other graphic products	2
5911-1/01		1
5911-1/02	Film production advertising	3
5911-1/99	Activities film, videos and television programs not specified elsewhere	1
5912-0/01	Dubbing Services	2
5912-0/02	Services in audiovisual production sound mixer	2
5912-0/99	Activities post-production film, videos and television programs not specified elsewhere	1
5913-8/00	Cinema distribution, video and television programs	1
5914-6/00	Activities Cinema Display	3
5920-1/00	Activities Sound recording and music publishing	2
6010-1/00	Radio activity	1
6021-7/00	Activities of broadcast television	3

NCEA 2.0	Description	Aliquot
6022-5/01	Programmers	3
6022-5/02	Activities related to pay television, except for programmers	3
6110-8/01	Services switched telephone network - PSTN	2
6110-8/02	Services telecommunications transport networks - SRTT	2
6110-8/03	Multimedia communication services - SCM	2
6110-8/99	Wired telecommunications services NES	3
6120-5/01	Mobile phone	2
6120-5/02	Specialized mobile service - SME	3
6120-5/99	Wireless telecommunications services NES	1
6130-2/00	Telecommunications Satellite	1
6141-8/00	Operators pay-TV cable	3
6142-6/00	Pay-TV operators microwave	2
6143-4/00	Operators pay television satellite	3
6190-6/01	Providers access to communications networks	3
6190-6/02	Providers of voice over internet protocol - VOIP	2
6190-6/99	Other telecommunications activities not specified elsewhere	2
6201-5/00	Development of custom computer programs	1

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
6202-3/00	Development and licensing of customizable computer programs	2
6203-1/00	Development and licensing of software non-customizable	1
6204-0/00	Consulting in information technology	2
6209-1/00	Technical support, maintenance and other services in information technology	2
6311-9/00	Data processing, service providers and application hosting services on the Internet	2
6319-4/00	Portals, content providers and other information services on the Internet	1
6391-7/00	News agencies	2
6399-2/00	Other activities to provide information services not specified elsewhere	3
6410-7/00	Central Bank	1
6421-2/00	Commercial Banks	2
6422-1/00	Multiple banks with commercial portfolios	3
6423-9/00	Savings	2
6424-7/01	Cooperative banks	1
6424-7/02	Central credit unions	1
6424-7/03	Credit unions	2
6424-7/04	Rural credit cooperatives	1
6431-0/00	Multiple banks, without commercial	1

NCEA 2.0	Description	Aliquot
6432-8/00	Investment Banking	1
6433-6/00	Development Banks	2
6434-4/00	Funding agencies	1
6435-2/01	Mortgage companies	1
6435-2/02	Savings and loan associations	1
6435-2/03	Mortgage companies	1
6436-1/00	Credit, finance and investment - financial	1
6437-9/00	Credit companies microentrepreneur	1
6440-9/00	Leasing	1
6450-6/00	Capitalization companies	3
6461-1/00	Holdings of financial institutions	2
6462-0/00	Holdings of non-financial institutions	3
6463-8/00	Other holding companies, except holdings	2
6470-1/01	Investment funds, except pension and real estate	1
6470-1/02	Pension investment funds	1
6470-1/03	Real estate investment trusts	1
6491-3/00	Societies to promote market - factoring	1

Tratado de Toxicologia Ocupacional

NCEA 2.0	Description	Aliquot
6492-1/00	Loan securitization	3
6493-0/00	Management of consortia for the acquisition of property and rights	2
6499-9/01	Investment clubs	1
6499-9/02	Investment companies	1
6499-9/03	Credit guarantee fund	1
6499-9/04	Cases of corporate finance	1
6499-9/05	Granting of credit for OSCIP	1
6499-9/99	Other financial service activities not specified elsewhere	1
6511-1/01	Life Insurance	1
6511-1/02	Plans for state funeral	2
6512-0/00	Non-life insurance	2
6520-1/00	Health insurance	1
6530-8/00	Reinsurance	2
6541-3/00	Pension fund system	1
6542-1/00	Open private pension	1
6550-2/00	Health Plans	2
6611-8/01	Stock exchange	1

NCEA 2.0	Description	Aliquot
6611-8/02	Commodities exchange	1
6611-8/03	Futures and commodities exchange	1
6611-8/04	Administration of OTC markets organized	2
6612-6/01	Brokerage Securities	1
6612-6/02	Distribution of securities	1
6612-6/03	Exchange brokers	1
6612-6/04	Brokerage commodity contracts	1
6612-6/05	Agents in financial investments	2
6613-4/00	Administration of credit cards	2
6619-3/01	Settlement and custody services	1
6619-3/02	Corresponding financial institutions	2
6619-3/03	Representatives of foreign banks	1
6619-3/04	ATMs	1
6619-3/05	Operators of debit cards	1
6619-3/99	Other activities auxiliary to financial services not specified elsewhere	2
6621-5/01	Experts and insurance assessors	1
6621-5/02	Audit and actuarial consulting	1

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
6622-3/00	Brokers, insurance, pension plans and health	1
6629-1/00	Activities auxiliary to insurance, pension and health plans not previously specified	2
6630-4/00	Fund management activities by contract or commission	2
6810-2/01	Buying and selling of own property	3
6810-2/02	Own rental property	2
6821-8/01	Brokerage in sale and property valuation	2
6821-8/02	Renting real estate brokerage	2
6822-6/00	Management and administration of real estate	2
6911-7/01	Legal services	1
6911-7/02	Activities auxiliary to justice	1
6911-7/03	Industrial property agent	1
6912-5/00	Notary Public	1
6920-6/01	Accounting activities	1
6920-6/02	Consulting and audit accounting and tax	2
7020-4/00	Consulting practice in business management, but specific technical advice	2
7111-1/00	Architecture services	3
7112-0/00	Engineering Services	3

NCEA 2.0	Description	Aliquot
7119-7/01	Mapping services, surveying and geodesy	2
7119-7/02	Geological activities	3
7119-7/03	Technical drawing services related to architecture and engineering	2
7119-7/04	Services of technical expertise related to work safety	1
7119-7/99	Technical activities related to engineering and architecture not previously specified	2
7120-1/00	Tests and technical analysis	1
7210-0/00	Research and experimental development on natural sciences and	2
7220-7/00	Research and experimental development on social sciences and humanities	1
7311-4/00	Advertising agencies	1
7312-2/00	Assemblage of spaces for advertising, except in the media	3
7319-0/01	Creation of stands for exhibitions	2
7319-0/02	Sales promotion	3
7319-0/03	Direct Marketing	3
7319-0/04	Advertising Consulting	2
7319-0/99	Other advertising activities not specified elsewhere	2
7320-3/00	Market research and public opinion	3
7410-2/01	Design	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
7410-2/02	Interior Design	3
7420-0/01	Activities of production photographs, except aerial and underwater	2
7420-0/02	Activities of production of aerial photographs and underwater	2
7420-0/03	Photo labs	2
7420-0/04	Footage of festivals and events	2
7420-0/05	Microfilming services	3
7490-1/01	Translation services, interpretation and similar	3
7490-1/02	Divers and dive	3
7490-1/03	Agronomy services and advice to agricultural and livestock activities	3
7490-1/04	Intermediation activities and agency services and business in general, except real estate	2
7490-1/05	Agency professionals for sports, cultural and artistic	3
7490-1/99	Other professional, scientific and technical expertise not previously specified	2
7500-1/00	Veterinary Activities	2
7711-0/00	Car rental without drivers	2
7719-5/01	Leasing of ships without crew, except for recreational purposes	2
7719-5/02	Leasing of aircraft without crew	3
7719-5/99	Renting of other transport NES without driver	3

NCEA 2.0	Description	Aliquot
7721-7/00	Equipment rental recreational and sports	2
7722-5/00	Rental of videotapes, DVDs and similar	3
7723-3/00	Rental objects of clothing, jewelry and accessories	2
7729-2/01	Rental of gaming devices	3
7729-2/02	Rental furniture, appliances and household appliances and personal; musical instruments	3
7729-2/03	Rental of medical	1
7729-2/99	Hire of other personal and household goods NES	3
7731-4/00	Hire of agricultural machinery and equipment without operator	3
7732-2/01	Rental of construction machinery and equipment without operator, except scaffolding	3
7732-2/02	Hire of scaffolding	3
7733-1/00	Rental of machinery and office equipment	1
7739-0/01	Rental of machinery and equipment for the extraction of minerals and petroleum, without operator	1
7739-0/02	Equipment rentals scientific, medical, no operator	3
7739-0/03	Rent a stage, roof and other structures for temporary use, except scaffolding	3
7739-0/99	Rent for machinery and commercial equipment and industrial NES without operator	3
7740-3/00	Management of intangible non-financial	1
7810-8/00	Selection and assemblage of manpower	3

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
7820-5/00	Hiring of manpower temporary	3
7830-2/00	Supply and human resources management for third parties	2
7911-2/00	Travel Agencies	1
7912-1/00	Tour Operators	1
7990-2/00	Reservation services and other tourism services NES	1
8011-1/01	Surveillance activities and private security	3
8011-1/02	Service training of guard dogs	2
8012-9/00	Activities of the transport of valuables	3
8020-0/00	Activities monitoring security systems	3
8030-7/00	Activities of Private Investigation	2
8111-7/00	Combined services to support the buildings, except building condos	3
8112-5/00	Condominium building	2
8121-4/00	Clean buildings and dwellings	3
8122-2/00	Immunization and control of urban pests	3
8129-0/00	Cleanup activities not specified elsewhere	3
8130-3/00	Landscaping activities	3
8211-3/00	Combined services of office and administrative support	2

NCEA 2.0	Description	Aliquot
8219-9/01	Photocopies	1
8219-9/99	Document preparation and specialized administrative support NES	3
8220-2/00	Telemarketing activities	3
8230-0/01	Services organization of fairs, congresses, exhibitions and parties	3
8230-0/02	Holiday parties and events	1
8291-1/00	Activities of collection and registration information	2
8292-0/00	Bottling and packaging under contract	3
8299-7/01	Measuring consumption of electricity, gas and water	3
8299-7/02	Issuance of food vouchers, transportation vouchers and similar	1
8299-7/03	Recording services stamps, except clothing	2
8299-7/04	Independent Auctioneers	2
8299-7/05	Fundraising services under contract	2
8299-7/06	Lottery	2
8299-7/07	Halls Internet Access	2
8299-7/99	Other activities of services mainly to companies not previously specified	2
8411-6/00	Administration of the State	2
8412-4/00	Regulation of activities in health, education, cultural services and other social services	1

Tratado de Toxicologia Ocupacional

NCEA 2.0	Description	Aliquot
8413-2/00	Regulation of economic activities	2
8421-3/00	Foreign relations	1
8422-1/00	Defense	1
8423-0/00	Justice	1
8424-8/00	Public order and safety	2
8425-6/00	Civil Defence	1
8430-2/00	Compulsory social security	1
8511-2/00	Infant education - nursery	2
8512-1/00	Infant education - pre-school	1
8513-9/00	Basic education	1
8520-1/00	High school	1
8531-7/00	Higher education - graduate	1
8532-5/00	Higher education - undergraduate and postgraduate	1
8533-3/00	Higher education - post-graduate and extension	1
8541-4/00	Technical professional education	1
8542-2/00	Professional education technology	2
8550-3/01	Administration of school boxes	1

NCEA 2.0	Description	Aliquot
8550-3/02	Activities to support education, except school boxes	2
8591-1/00	School sports	2
8592-9/01	Teaching dance	1
8592-9/02	School of performing arts, but dance	1
8592-9/03	Music education	1
8592-9/99	Teaching art and culture not previously specified	1
8593-7/00	Language Instruction	1
8599-6/01	Driver training	1
8599-6/02	Pilot Courses	3
8599-6/03	Computer training	1
8599-6/04	Training in professional and managerial development	1
8599-6/05	Preparatory courses for competitions	1
8599-6/99	Other teaching activities not specified elsewhere	2
8610-1/01	Activities of patient care, except emergency and emergency care units	2
8610-1/02	Activities of care in emergency rooms and hospitals for emergency care	2
8621-6/01	Mobile ICU	2
8621-6/02	Mobile services for emergency care, except for mobile ICU	2

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
8622-4/00	Removal services patients, except for mobile services in emergency care	2
8630-5/01	Activity resources for medical outpatient surgical procedures	1
8630-5/02	Medical practice with outpatient resources for laboratory testing	2
8630-5/03	Activity restricted to outpatient medical consultations	1
8630-5/04	Activity with resources for dental surgical procedures	1
8630-5/05	Activity without resources for dental surgical procedures	1
8630-5/06	Immunization services and human immunization	1
8630-5/07	Activities assisted human reproduction	2
8630-5/99	Outpatient care activities not specified elsewhere	2
8640-2/01	Pathology laboratories and cytological	2
8640-2/02	Clinical laboratories	2
8640-2/03	Dialysis units and nephrology	2
8640-2/04	Services tomography	1
8640-2/05	Diagnostic imaging services with the use of ionizing radiation, except CT	2
8640-2/06	MRI Services	2
8640-2/07	Diagnostic imaging services without using ionizing radiation, but magnetic resonance	1
8640-2/08	Diagnostic services by record chart - ECG, EEG and other tests similar	3

NCEA 2.0	Description	Aliquot
8640-2/09	Diagnostic services by optical methods - endoscopy and other tests similar	2
8640-2/10	Chemotherapy Services	2
8640-2/11	Radiotherapy services	2
8640-2/12	Services hemotherapy	1
8640-2/13	Lithotripsy services	1
8640-2/14	Services of banks of cells and tissues	1
8640-2/99	Activities services diagnostic testing and treatment not specified elsewhere	2
8650-0/01	Nursing activities	1
8650-0/02	Activities of nutrition professionals	3
8650-0/03	Activities of psychology and psychoanalysis	1
8650-0/04	Physical therapy activities	1
8650-0/05	Activities of occupational therapy	2
8650-0/06	Speech Therapy Activities	1
8650-0/07	Activities therapy enteral and parenteral nutrition	1
8650-0/99	Activities of health professionals not previously specified	2
8660-7/00	Activities to support health management	2
8690-9/01	Activities complementary and integrative practices in human health	2

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
8690-9/02	Activities of human milk bank	1
8690-9/99	Other activities focus on human health is not specified elsewhere	2
8711-5/01	Clinics and geriatrics	2
8711-5/02	Long-stay institutions for the elderly	2
8711-5/03	Activities of assistance to disabled people, immunosuppressed and convalescent	1
8711-5/04	Support centers for cancer patients and AIDS patients	3
8711-5/05	Residential developments for elderly and disabled	2
8712-3/00	Activities providing infrastructure support and assistance to patients at home	2
8720-4/01	Activity centers for psychosocial	1
8720-4/99	Activities of psychosocial and health care for persons with mental disorders, mental retardation and substance abuse not previously specified	2
8730-1/01	Orphanages	2
8730-1/02	Hostel care	2
8730-1/99	Welfare activities provided in group and private residences not specified elsewhere	2
8800-6/00	Social services without accommodation	2
9001-9/01	Theatrical production	1
9001-9/02	Production Music	2
9001-9/03	Production of dancing	2

NCEA 2.0	Description	Aliquot
9001-9/04	Production of Circus performers, puppets and similar	1
9001-9/05	Production of shows rodeos, rodeos and similar	3
9001-9/06	Activities of sound and lighting	1
9001-9/99	Arts, entertainment and additional activities not specified elsewhere	3
9002-7/01	Activities of artists, independent journalists and writers	1
9002-7/02	Restoration works of art	1
9003-5/00	Space management for the performing arts, performances and other artistic activities	3
9101-5/00	Activities of libraries and archives	2
9102-3/01	Activities and operation of museums and places of historical buildings and attractions like	1
9102-3/02	Restoration and conservation of historic buildings and places	1
9103-1/00	Activities of botanical gardens, zoos, national parks, ecological reserves and protected areas	2
9200-3/01	Bingo halls	1
9200-3/02	Exploration of betting on horse racing	2
9200-3/99	Exploration of gambling and betting NES	1
9311-5/00	Management of sports facilities	2
9312-3/00	Social clubs, sports and similar	2
9313-1/00	Fitness activities	1

Tratado de Toxicología Ocupacional

NCEA 2.0	Description	Aliquot
9319-1/01	Production and promotion of sporting events	2
9319-1/99	Other sporting activities not specified elsewhere	2
9321-2/00	Amusement parks and theme parks	2
9329-8/01	Nightclubs, discos, dance halls and similar	1
9329-8/02	Exploration of bowling	3
9329-8/03	Operation of games of snooker, billiards and similar	1
9329-8/04	Operation of recreational games	3
9329-8/99	Other leisure and recreational activities not specified elsewhere	2
9411-1/00	Activities of membership organizations of employers and business	3
9412-0/00	Activities of professional membership organizations	3
9420-1/00	Activities of trade unions	2
9430-8/00	Activities of associations of social advocacy	2
9491-0/00	Activities of religious organizations	2
9492-8/00	Activities of political organizations	1
9493-6/00	Activities of membership organizations related to culture and art	2
9499-5/00	Associational activities not specified elsewhere	2
9511-8/00	Repair and maintenance of computers and peripheral equipment	3

NCEA 2.0	Description	Aliquot
9512-6/00	Repair and maintenance of communications equipment	2
9521-5/00	Repair and maintenance of electronic equipment for personal and household	3
9529-1/01	Repair of footwear, handbags and travel goods	1
9529-1/02	Locksmiths	3
9529-1/03	Repair of watches	1
9529-1/04	Repair of bicycles, tricycles and other non-motor vehicles	3
9529-1/05	Repair of furniture items	2
9529-1/06	Jewelry Repair	2
9529-1/99	Repair and maintenance of other objects and personal and household equipment NES	3
9601-7/01	Laundry	3
9601-7/02	Dye	3
9601-7/03	Towel	3
9602-5/01	Hairdressers	2
9602-5/02	Other activities of beauty treatment	2
9603-3/01	Management and maintenance of cemeteries	3
9603-3/02	Cremation Services	2
9603-3/03	Burial services	2

Tratado de Toxicologia Ocupacional

NCEA 2.0	Description	Aliquot
9603-3/04	Funeral Services	2
9603-3/05	Services somatoconservação	3
9603-3/99	Activities Funeral and related services NES	3
9609-2/01	Esthetic clinics and similar	1
9609-2/02	Marriage agencies	3
9609-2/03	Accommodation, cleanliness and beautification of animals	2
9609-2/04	Operation of personal service machines driven by currency	1
9609-2/99	Other activities of personal services not specified elsewhere	2
9700-5/00	Domestic Services	2
9900-8/00	International organizations and other extraterritorial institutions	1

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